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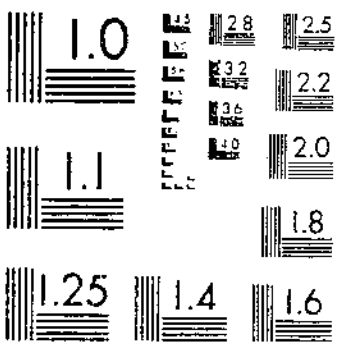


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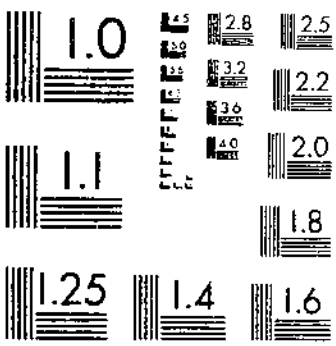


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# RELATIVE EFFICIENCY OF VARIOUS GENETIC MECHANISMS FOR SUPPRESSION OF INSECT POPULATIONS

By E. F. Knipling and W. Klassen

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# RELATIVE EFFICIENCY OF VARIOUS GENETIC MECHANISMS FOR SUPPRESSION OF INSECT POPULATIONS

By E. F. Knipling, *collaborator*, and W. Klassen, *staff scientist*,
*National Program Staff, Agricultural Research Service*
*United States Department of Agriculture*

## SUMMARY

A theoretical appraisal was made of the relative efficiency of various genetic mechanisms of suppression when applied to natural pest populations. These appraisals were based on the release of genetically modified males or males plus females in most cases when they outnumbered the wild population by 9:1. The effects of a single release or of releases for three successive generations were calculated. The theoretical suppression effect is compared with that expected from the same rate of release of insects sterilized conventionally (dominant lethal mutations). The genetic mechanisms considered include: Compound chromosomes, cytoplasmic incompatibility, hybrid sterility, imbalanced sex determining factors, inherited partial sterility, chromosomal translocations, inherited hybrid male sterility, sex-ratio distortion, recessive lethals, and conditional lethals, both dominant and recessive.

On the basis of comparable competitiveness, several genetic mechanisms would be more effective than the release of sterile males. The most efficient mechanisms based on these theoretical appraisals would be: Meiotic drive coupled with a dominant conditional lethal, a compound chromosome coupled with a dominant conditional lethal, inherited hybrid male sterility, and inherited partial sterility. Other mechanisms such as translocations and several dominant conditional lethals in a single strain may also be more effective than conventional sterility. Other mechanisms such as imbalanced sex determinants, as occur in the gypsy moth, and sex-ratio distortions, as occur in the house fly, seem to have little or no suppression advantage over the release of sterile males but could be more effective because of increased competitiveness.

While the suppressive effect of various genetic mechanisms will depend on competitiveness and physical fitness of the released strain in relation to the normal wild strain in the environment, a theoretical appraisal by the procedures employed provides an opportunity to identify genetic mechanisms that offer maximum potential suppressive action, if appropriate genetic alternatives can be developed in strains suitable for release.

## INTRODUCTION

Scientists are increasingly interested in the autocidal method of pest suppression utilizing various genetic techniques. This interest is due to several factors. First, the sterile-male technique has been shown to be of practical value and is being employed in the suppression of the screwworm (*Cochliomyia hominivorax* (Coqueral)), pink bollworm (*Pectinophora gossypiella* (Saunders)), Mexican fruit fly (*Anastrepha ludens* (Loew)), and Mediterranean fruit fly (*Ceratitis capitata* Wiedemann)). Encouraging results have been obtained against a number of other important insects including boll weevil (*Anthonomus grandis* Boheman), tsetse flies, *Glossina* spp., codling moth (*Carpocapsa pomonella* (Linn.)), certain mosquitoes, several other species of tropical fruit flies, and a number of other pests. Many scientists appear to appreciate the role that the technique could play, especially in integrated pest management systems for key insect pests. Secondly, there is the general realization that new ecologically acceptable strategies are needed to cope with insect pest problems. The genetic approach offers a highly selective means of control. Thirdly, geneticists and entomologists are discovering or engineering new genetic suppression mechanisms that promise to be more effective than the induced sterility procedure, which has received most of the attention in research up to the present time. Fourthly, important strides have been made in developing mass rearing and quality control technology. In addition, the capability to identify important behavioral and physiological features of mass-reared and released insects has increased immensely, as has the capability to analyze problems with field performance of released insects.

Many potential genetic suppression mechanisms have been known for some time and many of them are being investigated, but relatively little attention has been given to an appraisal of the efficiency of various suppression mechanisms when applied to natural populations. This is the purpose of this publication. We recognize that the true effect various techniques will have in the suppression of pest populations cannot be determined until properly planned and properly executed experiments are conducted in the field. However, field experiments are difficult and costly. Research resources are totally inadequate to undertake the many field investigations that would be desirable. Based on the success of earlier predictive models of the sterile-male technique, however, much can be done through appropriate modeling

and calculating procedures, with or without computer programming, to appraise the potential for suppression that various known genetic mechanisms have. The results of appraisals herein reported show great differences in the potential efficiency of various genetic systems. The potential suppression that can result from various mechanisms to be described must be related to the dynamics of the target insect population as we would expect it to occur in a natural environment.

We hope that our findings in this study will not only stimulate further fundamental investigations on new genetic suppression mechanisms, but also serve as a guide for practical laboratory and field investigations designed to develop the most promising genetic principles for practical insect population suppression.

## THE NATURE OF ACTION OF VARIOUS GENETIC MECHANISMS

Genetic mechanisms that are potentially useful for insect population suppression and the population replacement may be classified in three categories depending on the degree of involvement and the speed and duration of action of the genetic material introduced.

Category A involves no infusion of genetic material, and the impact of the mechanisms does not extent beyond one generation. The mechanisms involved include: (1) Dominant lethal mutations (the basis of the sterile insect release method first used against the screwworm); (2) compound chromosomes (provided that individuals of one sex only are released); (3) cytoplasmic incompatibility (provided that males only are released); and (4) hybrid sterility (provided that $F_1$ hybrid sterile males only are released).

Category B involves mechanisms that have an impact beginning in the first generation and that extend for a number of additional generations. In this category the altered and released germ plasm is involved in reproduction. This category includes: (1) Inherited partial sterility in species with holokinetic chromosomes; (2) chromosome translocations (provided that male heterozygotes only are released); (3) compound chromosomes (provided that both sexes are released); (4) inherited hybrid male sterility (provided that both sexes of $F_1$ generation are released); and (5) cytoplasmic incompatibility (provided that both sexes are released).

The mechanisms in category C have no impact until after the first generation following the release. Thus, genes are introduced into the wild population which are expressed in later generations.

The genetic mechanisms in category C include: (1) Recessive genes for nonvector capacity, insecticide susceptibility, and preference for economically unimportant hosts; (2) recessive lethals; (3) conditional lethals; (4) sex-ratio distortion through mechanisms that produce mostly male progeny; (5) sex-ratio distortion through the use of unisexual recessive lethals, that is, those that are sex linked and lethal in double dose only; (6) meiotic drive in males or nonrandum disjunction in females (of course, these drive mechanisms must be linked to any one of the mechanisms C1 to C5 inclusive); (7) homozygous chromosome translocations; and (8) hybrid sterility involving the release of both sexes—the male outcrosses are sterile, and female outcrosses to normal males are partially or fully fertile.

An understanding of the basis of the various genetic mechanisms is helpful in modeling their impact on wild populations and in identifying their limitations. However, to appraise the impact representative normal trends of insect population must be established. The following account is intended to explain the nature of some of the presently known mechanisms. Later, we will compare their potential impact on insect population trends. While references will be made to certain pertinent publications, a thorough review of the already extensive literature on genetic mechanisms of insect control is beyond the scope of this publication.

## Sterility Caused by Dominant Lethal Mutations

Ionizing radiation and chemosterilants sterilize insects by causing lethal changes in the hereditary material of the germ cells. These lethal changes are designated as dominant lethal mutations. By definition only one of the two germ cells which unite at fertilization to form the zygote need carry the dominant lethal mutation to kill the individual that develops from the zygote. Thus, a dominant lethal mutation does not kill the germ cell in which it is induced. For example, if the mutation is induced in a spermatid, it will pass into a mature sperm which fertilizes the egg. The dominant lethal mutation expresses itself in the embryo or at a later stage. Mutagenic agents induce these mutations most frequently by breaking chromosomes. A thorough discussion of the induction and nature of dominant lethal mutations is found in LaChance and others (*24, pp. 99-157*) and in LaChance and Riemann (*25*).[1] Excellent discussions of lethal

[1] Italic numbers in parentheses refer to Literature Cited, p. 53.

chromosome aberrations may be found in Muller (*30, pp. 851-478*) and in Smith and von Borstel (*38*).

The use of contrived dominant lethality was suggested by Smith and von Borstel (*38*) to circumvent two undesirable direct effects of mutagenic agents on insects that are released for population suppression. In some species, such as a boll weevil, mutagenic agents used to induce sterility inflict considerable somatic damage —which contributes to reduced competitiveness. In addition, the sterilizing treatment must destroy all primary germ cells so that the insects will not recover fertility. (This would not apply to Lepidoptera since spermatogenesis does not occur in the adult.) Therefore, males sterilized with mutagenic agents can transfer sperm in a limited number of matings only. Matings in nature will be limited to the average matings that females will accept, but in mass rearing and sterilization many matings may occur under crowded conditions. These limitations may be circumvented by developing two separate strains, each homozygous for several different chromosomal translocations. When males of one strain are mated to females of the other strain, progeny are produced that are heterozygous for all the translocations. Smith and von Borstel (*38*) suggested that if each strain contained three different translocations, over 98 percent of the gametes of the heterozygote would be unbalanced genetically. These sterile heretozygotes should be normal, and they would produce normal quantities of gametes.

## Translocations and Inherited Partial Sterility

Sometimes when a mutagen breaks several chromosomes in one cell, a fragment of one chromosome attaches to a fragment of another chromosome to form a translocation; if two nonhomologous chromosomes interchange, they form a reciprocal translocation. This is the most common type of translocation. In translocation homozygotes, the chromosomes should behave like standard chromosomes during meiosis. However, in translocation heterozygotes, the chromosomes form a crosslike configuration during prophase I because of the close pairing of homologous parts. At a later stage, the crosslike figure opens up into a 4-membered ring. Sometimes this ring twists on itself to form a "figure 8." When this happens, alternate members of the configuration go to the same pole when the cell divides. The daughter nuclei then have complete chromosomal complements, with half of them carrying reciprocal translocation chromosomes. When adjacent chromosomes in the ring configuration go to the same

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pole, the daughter nuclei receive duplications and deficiencies. When many chromosomes become involved in a multiple reciprocal translocation, all the chromosomes involved form a ring or a chain at meiosis.

A most important consequence of reciprocal translocation is the partial sterility of many translocation heterozygotes. In an insect heterozygous for a single reciprocal translocation, chromosomes from the meiotic translocation figure may pass two by two in random assortment to opposite poles. Here, two-thirds of the resulting gametes would have chromosomal duplications and deficiencies; these constitute dominant lethal mutations. In reality completely random assortment of translocated chromosomes does not occur. Indeed, a single translocation in an individual can cause the death of 20 to 80 percent of its progeny (Waterhouse and others (*47*)). In this paper, however, we will assume that about half the gametes contain duplication and deficiencies.

If two chromosomes are involved in a translocation, we would expect roughly 50 percent hatch of the eggs from a mating that involves a translocation heterozygote; if the individual possesses two translocations in heterozygous form, then we might expect roughly 25 percent hatch; and with three translocations, we might expect 12.5 percent hatch, and so on. Moreover, half the progeny produced by a translocation heterozygote would bear the translocation, and these progeny would produce some sperm bearing contrived dominant lethal mutations. This is a bonus effect that is not obtained by the use of fully sterile insects. It will be shown that releases of insects bearing several translocations would have a more profound effect on a wild population than releases of fully sterile males. Another possible advantage is that insects bearing certain translocations may be fully competitive sexually, in contrast to fully sterile insects that may be damaged by the sterilizing treatment.

The use of translocations may take two forms. The first involves the development of a strain of insects homozygous for several translocations. A chemosterilant or radiation is used to break the chromosomes. Then, the progeny are examined cytologically for the presence of translocations (*4*) or by progeny tests using genetic marker strains to determine changes in linkage relationships. The other approach involves the exposure of insects that possess diffuse centromeres to a dose of a chromosome-breaking agent that induces partial sterility only (*31; 32, pp. 391-403; 33, pp. 99-111*).

In insects with diffuse or multiple centromeres, acentric frag-

ments are not produced by chromosome breaks; thus chromosome fragments are not lost as a result of chromosome breakage. The broken ends of a chromosome may rejoin to restitute the original chromosome, or they may join to the unbroken ends of nonhomologus chromosomes in reciprocal or other types of translocations. When species whose chromosomes possess just one centromere are treated with a chromosome-breaking mutagen, translocations are induced in a small percentage of the cells. A higher translocation frequency is not realized because most acentric fragments are lost before they have an opportunity to unite with centric fragments, and because dicentric isochromosomes are produced that form lethal bridges in most cells where chromosome breaks occur. However, in species whose chromosomes have diffuse centromeres, all fragments have attachments to the spindle fiber, and under moderate treatment it seems likely that they persist, rarely form isochromosomes, and unite with other fragments in translocation. Thus, it is possible to obtain several translocations in every gamete.

When the cabbage looper males are irradiated with 20 Krad of X-rays and mated to untreated virgins, approximately 20 percent of the eggs hatch, and these progeny are sterile or semisterile because they possess several translocations (*32, pp. 391-403*) and some fail to transmit eupyrene sperm. Extensive literature is available on translocations and inherited sterility relevant to population suppression. Publications with extensive bibliographies include those of Curtis and Hill (*5*), Smith and von Borstel (*38*), Wagoner (*42*), Wagoner and others (*45, pp. 183-197*), Waterhouse and others (*47*), and Whitten (*48*).

## Compound Chromosomes

According to Novitski (*34*) and by personal communication[2] there can be a number of variations in compound chromosomes. For this analysis we will consider that in the compound chromosome the left arm is identical to the right arm, that is, two homologous chromosomal arms are attached to the same centromere. Normally, heterologous arms are attached to one centromere. Compounds of the sex chromosomes and of the autosomes have been available in *Drosophila melanogaster* for a number of years. Foster and others (*9*) described special methods of constructing strains of insects with compound chromosomes. These authors,

[2] Novitski, E., Department of Biology, University of Oregon, Eugene, Oreg. 97403.

Childress (*2*) and Fitz-Earle and others (*8*) proposed that compound chromosomes could be used as transport mechanisms for introducing conditional lethals and other genes into a population.

For example, the normal arrangement of arms in a pair of homologous chromosomes is left arm-centromere-right arm (L-R), and left arm-centromere-right arm. By appropriate means these arms can be arranged as follows: Left arm-centromere-left arm (L-L) and right arm-centromere-right arm (R-R). Four types of gametes are formed by a compound chromosome strain: L-L, R-R, L-L plus R-R, and no compound chromosome (nullisomic). In *Drosophila melanogaster* these gametes are formed by males and females in approximately equal proportions (fig. .). Therefore

| female gametes / male gametes | L—L | R—R | L—L and R—R | — |
|---|---|---|---|---|
| L—L | L—L<br>L—L<br>lethal | R—R<br>L—L | L—L<br>R—R<br>L—L<br>lethal | L—L<br>lethal |
| R—R | L—L<br>R—R | R—R<br>R—R<br>lethal | R—R<br>L—L<br>R—R<br>lethal | R—R<br>lethal |
| L—L and R—R | L—L<br>L—L<br>R—R<br>lethal | R—R<br>L—L<br>R—R<br>lethal | L—L<br>R—R<br>L—L<br>lethal | L—L<br>R—R |
| — | L—L<br>lethal | R—R<br>—<br>lethal | L—L<br>R—R | —<br>lethal |

FIGURE 1—Types of zygotes produced from a cross between parents both bearing compounded left and right arms for the same chromosome. Random segregation results in 16 zygotes of which 12 are genetically imbalanced and lethal. Directed segregation produces 4 zygotes of which 2 are genetically imbalanced and lethal.

such strains are about 25 percent fertile. Also, if a normal individual mates with a compound chromosome-bearing individual, all zygotes will be genetically imbalanced and therefore incapable of development. Therefore, if a normal population is overflooded with a strain bearing a compound chromosome, only two types of progeny will result; that is, those from the normal × normal cross and those from the compound × compound cross. Childress (*2*) pointed out that if a 25-percent fertile strain overfloods a normal strain in a ratio greater than 4:1, then, theoretically, the normal strain will be totally destroyed and replaced by the compound chromosome strain after a number of generations. Further, if the self-sterility of the release strain is 50 percent and if the normal strain is overflooded in a ratio exceeding 2:1, then the normal strain eventually will be totally replaced.

If the normal strain is replaced by the chromosome-bearing strain during the regular favorable season for survival (permissive conditions) but the compound chromosome strain cannot survive because of conditionally lethal characteristics during the subsequent unfavorable season (restrictive period), both strains should essentially disappear. This will be considered in a subsequent section.

If males only of a compound chromosome-bearing nondiapause strain were released, then the final impact on the target population would be identical to that produced by releasing sterile males. A compound chromosome-bearing strain could not be established in the ecosystem in the absence of compound chromosome-bearing females.

## Conditionally Lethal Mutations

The major handicap in the use of recessive lethal mutations for population suppression lies in the unavoidable use of release strains that are heterozygous for the mutation. Thus for every lethal allele that is introduced into the population, a normal allele is also introduced. This difficulty can be circumvented by the use of conditionally lethal mutations. Conditionally lethal mutations permit an organism to survive under certain conditions (permissive) but not under other conditions (restrictive). Temperature-sensitive lethals are an important class of conditional mutations that should be useful for suppressing pest species—whether or not a formal genetics has been erected (*4; 37, pp. 453-465; 38; 39; 45, pp. 183-197; 48*).[3] The inability to fly should be an im-

[3] Bartlett, A. C. Personal communication, Western Cotton Insects Laboratory, Phoenix, Ariz. 85040.

portant conditional lethal for insects that need to fly to reproduce successfully. Such a mutation in the boll weevil would express itself when the insects attempted to leave the cotton fields for hibernation or when leaving hibernation sites in search of host plants.

Another rich source of conditional lethals are found in adaptations of pest species to climate. Insects are adapted for survival in regions marked by climatic extremes during the annual cycle. These adaptations synchronize the insects' sensitive growing stages with seasons when food is present and freezing temperatures and droughts are absent. Further, these adaptations permit survival during periods of extreme cold and lack of water or food. Characteristically, insects survive adverse periods by entering diapause before the onset of an adverse period and by terminating diapause only when favorable conditions again prevail. Many pest species have wide ranges of distribution in which seasons vary greatly between localities. Therefore, diapause characteristics of local populations must vary accordingly, and the characteristics of local populations must be hereditary. To date most thought has been given to utilizing the inability to diapause. Other potential, conditional lethals are the inability to respond to the appropriate critical photoperiod, inadequate duration of diapause, and inability to develop sufficient cold hardiness (*16; 18, pp. 65-79; 19; 22; 23*).

## Hybrid Sterility

Hogan (*15*) pointed out that when an insect species has a wide geographic distribution with a wide diversity of habitats, races tend to develop with characteristics favoring survival in each type of environment. The adaptations of each race are mainly physiological and derive from mutations with survival value which are not transmitted to other races because of reproductive isolation. By such processes new species may be developed.

Allopatric populations may accumulate sufficient physiological differences to prevent the development of embryos when hybrid matings occur in interconnected populations. Indeed, Vanderplanck (*41*) found that individuals of the allopatric tsetse fly species, *Glossina swynnertoni* and *Glossina morsitans*, would intermate at random but that the crosses yielded few or no progeny. Vanderplanck (*41*) suggested that control of *G. swynnertoni* must be achieved by mass releases of *G. morsitans* in its population, and vice versa. The numerically superior species would sterilize the numerically inferior species. Once the indigenous species had been

eliminated, the introduced species would fail to survive indefinitely because it would not be adequately adapted to the environment.

The *Anopheles gambiae* complex consists of five separate species that are geographically isolated. Matings between the various species in either direction occur readily to produce sterile hybrid males and fertile hybrid females. Sterility in males appears to be caused by lack of complete homology of chromosomes which cause extensive asynapsis. As the result of asynapsis, the orderly separation of chromosomes at meiosis does not occur, so that the sperm do not receive the proper number of chromosomes. Females are unaffected because they pass off aberrant chromosomes in the polar bodies. Testes of hybrid males are not definitively developed and are often devoid of sperm, yet the sexual behavior of hybrid males may be enhanced by heterosis. Indeed, hybrid sterile males appear to be suited for use in population suppression programs (*6, pp. 211-250*).

Another example of hybrid sterility between allopatric races was discovered in the gypsy moth, *Porthetria dispar*, by Goldschmidt (*11; 12, pp. 430-431*). Goldschmidt found that males of strains from northern Japan when crossed to females from southern Japan or from Europe produced fertile sons, but in place of normal daughters, sterile intersexes appeared. Further, if these $F_1$ males were backcrossed to females from southern Japan or Europe, fertile sons and sterile intersexes were produced. Initially Goldschmidt had suggested that sex was determined by Mendelian factors, then in his 1915 paper Goldschmidt (*10*) presented data that led him to believe that both Mendelian and cytoplasmic factors are involved. Goldschmidt suggested that the X chromosome has male-determining factors that must be properly balanced by female-determining factors. Goldschmidt believed that the male—having two X chromosomes—has two doses of male-producing factors and has one dose of female-determining factors in the cytoplasm.

Further, Goldschmidt believed that the female has one X chromosome providing only one dose of male-determining factors that is balanced against the one dose of female-determining factors. However, the "strength" of these factors varies between geographic races. In particular, males from northern Japan have strong male-producing X chromosome factors while males from southern Japan and Europe have weak male-determining factors. In each strain, the female-producing factors have the appropriate "strength" to produce a male if the embryo cells have two X chromosomes and to produce a female if these cells have one X

chromosome. Interracial crosses bring together improperly balanced male- and female-determining factors so that intersexes may be produced.

Based on this interpretation, Downes (*7*) proposed that strong-race males could be used to suppress the weak race in North America. However, Goldschmidt (*12*) withdrew some of his earlier conclusions and suggested that female sex determination is not cytoplasic but dependent on the Y chromosome. Further, he indicated a phenomenon that could hinder the suppression of the gypsy moth by the method of Downes. Evidently, a strong male sometimes may appear with two X chromosomes and a Y chromosome. Such males occasionally may produce sperm bearing a Y chromosome and produce fertile daughters when mated to a female of the weak race. Thus, the mass release of strong males only into North American populations could result in the transmission of the Japanese Y chromosome to produce fertile hybrid females. Such females would be weak X, strong Y. These females would mate to the released Japanese males to produce a new generation with females strong X, strong Y (similar to Japanese females) and weak X, strong X males. The strong Y chromosome could be established and displace the weak Y chromosome in the American strain. Here, further releases of strong males would be futile, and other means would have to be used to suppress the local population. Of course, this technique in itself should not discourage work to develop the method of Downes, but such an effort should include research to actually establish the mechanism of sex determination in the pest. At the outset an effort should be made to determine whether the gypsy moth has a Y chromosome.

Another potentially useful example of hybrid sterility was discovered by Graham and others (*14*) by crossing the cattle ticks, *Boophilus annulatus* and *B. microplus*. Cross-mated females produce normal numbers of eggs with normal hatch, and the $F_1$ progeny are normal in vigor and longevity. However, the hybrid sons are more than 99 percent sterile, and the hybrid daughters are partially sterile. Graham and others (*14*) suggest that these hybrid males might be used for genetic control of either species, although such an attempt would have to be preceded by additional research. As we will consider later in discussing inherited hybrid sterility, if the hybrid sterility persists in backcrosses, such sterility mechanism could prove useful as a means of suppressing low-level, incipient infestations.

## Cytoplasmic Incompatibility

Cytoplasmic incompatibility has been studied most extensively in *Culex* mosquitoes, is known to occur in *Aedes scutellaris* mosquitoes, and has not been found in houseflies. When males of a given strain of *C. pipiens* are crossed with females of a strain from a different geographical area, the number of progeny may be normal, small, or zero. These crossed strains are referred to as compatible, partially compatible, or incompatible, respectively. Partial compatibility and incompatibility are caused by the blockage of the sperm after it enters the egg and before its nucleus can participate in forming a diploid zygote. Nevertheless, cleavage divisions and embryogenesis may occur; yet, such embryos die before hatching because they are haploid. Cytoplasmic incompatibility is a naturally occurring phenomenon and has been used to totally suppress *Culex* populations. Laven and Aslamkhan (*27*) suggest that the combination of cytoplasmic incompatibility with translocation heterozygosity could be a powerful, suppressive system. Yen and Barr (*49*) found a rickettsialike microorganism, possibly *Wolbachia pipientis*, in eggs of *Culex* but not in the sperm. These authors suggest that various geographical strains of this micro-organism are well-adapted symbionts of the *Culex* mosquitoes in the areas where they are usually found. However, these transovarially transmitted symbionts are deleterious to the sperm of strains from other geographical areas.

## Meiotic Drive and Sex-Determination Abnormalities

Meiotic drive refers to the preferential recovery of one particular member of a pair of homologous chromosomes at meiosis, that is, wide deviation from the expected 1:1 ratio (*36*). For example, in the yellow fever mosquito, Craig and others (*3*) discovered an inherited factor, *Distorter*, which causes a male to produce a disproportionate number of sons. These high male ratios are the consequence of males with *Distorter* producing a normal complement of male-determining sperm and a deficient complement of female-determining sperm. Meiotic drive has been considered as a mechanism to carry desired deleterious genes into a wild population. Foster and others (*9*) postulate that such meiotic drive is closely associated with recessive sterility, thus preventing the fixation of the desired genes in the population.

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However, as shown by Novitski and Hanks (*35*), meiotic drive could involve many different biological bases and is not necessarily associated with recessive sterility. In female *Drosophila* nonrandom disjunction is a drive phenomenon associated with unequal crossing over, that is, the shorter chromatid is recovered much more frequently than the longer chromatid. Wagoner and others (*45, pp. 183-197*) suggest that a combination of a drive mechanism in the male and nonrandom disjunction in the female may prove to be an effective mechanism for population suppression.

In certain strains of houseflies dominant male-determining factors are found on autosomes 2, 3, and 5 (*43*). Thus, sex is determined by mechanisms in addition to the X and Y chromosomes. Further, in some housefly strains the Y chromosome appears to be replaced by an X chromosome. In some strains one or more dominant female-determining factors are present as well. Thus, strains have been synthesized in which the males, when outcrossed to females from wild strains, produce male progeny only (*29, pp. 38-397; 44; 45, pp. 183-197*). Wagoner and Johnson (*44*) suggest that the effect of releasing male-producing males into wild populations would be similar to that obtained with the sterile-male technique, with the exception that the use of male-producing males is attended with the presence of many male flies in the wild population. Wagoner and Johnson (*44*) also suggest that for sterile-male programs to suppress houseflies, males from a male-producing strain could be mated to females from a normal strain in the laboratory so that males only would be produced for sterilization and subsequent release. This procedure would cut rearing and handling costs in half.

## ESTABLISHMENT OF PARAMETERS FOR THE POPULATION MODELS

Population simulation models can be useful in appraising the relative efficiency of various genetic suppression mechanisms. Such models should reasonably reflect the population dynamics of insects that are good candidates for control by genetic means. In virtually all situations where insect control is likely to be feasible by genetic manipulation, the natural pest population must be at a low-density level for it to be practical to adequately overflood the natural population. Under such conditions the natural density-related suppression forces are generally at the lowest intensity. Thus, the natural population is likely to have nearly maximum innate capacity for increase for the particular environ-

ment in which it exists. This potential for natural increase must be nullified to cause a suppression of the population. The purpose of genetic control will be to either suppress a low natural population to an even lower level or to slow down the rate of reproduction enough to significantly delay population increase to economic threshold levels. The complete elimination of insect populations may be the goal in some circumstances, but complete elimination of populations usually will not be feasible or practical because of lack of isolation from immigrants. In such event the genetic method might still play a prominent role as a selective means of maintaining low populations or to delay the development of economic populations. In either event maximum efficiency can be expected only when pest populations are at very low-density levels (*17*).

The number of insects present in well-established natural pest populations is generally so high during periods of moderate- to high-density levels that it will be impractical to rear and release enough insects to achieve the desired suppression effect. On the other hand, many important pest species are present in relatively low numbers during periods of scarcity, or technology is available for reducing the numbers to low levels by chemical, cultural, or other means. Moreover, the technology on mass production of insects has advanced to a remarkable degree in the past decade, and it seems entirely feasible now to rear and release more genetically altered insects than might be expected in natural populations of many species during periods of scarcity or when purposely reduced. Further advances in this area can be expected. Developments to achieve maximum efficiency in the application of genetic suppression principles, as well as in the production of high-quality competitive insects, are equally important since practical use of the genetic principles hinges on success in both fields for any given pest.

This study was made to compare the relative potential efficiency of various genetic mechanisms. Thus, insofar as possible, all comparisons will be made using the same basic parameters. All the insects released are assumed to be fully competitive in mating with members of the natural population. They are distributed so as to have the same chance as the native insects for mating with members of the natural population. We recognize fully that insects released are likely to vary in their ability to compete for mates in a natural population. Also, we know that induced sterility by radiation or by chemical means, adversely affects the vigor of some species. In all probability, sterile hybrid insects

produced by crossing different species or races may not have the same ecological or mating behavior as the target species. Also, various genetic defects engineered into strains may adversely affect the vigor or behavior, or both, of the individuals released. Nevertheless, it is important to appraise and recognize the inherent capability for suppression that different genetic mechanisms have. This can be important in establishing priorities for research on various types of genetic mechanisms both in the laboratory and in the field. The actual performance of insects released in the field is the final test for any genetic suppression mechanisms, but such an evaluation is outside the scope of the study reported here.

Unless otherwise specified, the relative suppression potential for different genetic mechanisms will be calculated on the basis of two release systems: (1) The release of insects in one generation (parent) only; (2) the release of insects for three successive generations (parent, $F_1$, and $F_2$ generations).

To compare the relative efficiency of the different genetic systems, most of the releases are programed at the same rate each generation. This does not mean that the release rate used is necessarily the optimal rate, but calculations based on constant variables will facilitate an appraisal of the relative impact of the different systems of suppression. All hypothetical natural populations will start at the level of 1,000 females and 1,000 males. The genetically altered insects will be released at the rate of 9,000 males each generation when releases are programed. This will provide an initial ratio of nine released males to one native male in the first generation. The ratio of released to native insects will subsequently vary depending on the number in the natural population.

In some models males only will be released; in others, both sexes will be released. If females are released, they will outnumber the native females by a ratio of 9:1 in the first generation.

The natural population and all insects capable of fertile matings are assumed to increase fivefold each generation. This means that a fertile mating will result in 10 progeny. No adjustments are made in the rate of increase of populations due to a relaxation of density-dependent suppression forces. In genetic control, we are generally dealing with low populations and density-dependent suppression forces are not likely to vary substantially within the population range that will be maintained in practical control. The sexes of all progeny are assumed to be equal except where the genetic mechanisms results in the death of one sex. The female insects are monogamous. However, if sperm from the

released insects are fully competitive in all respects with those from native males, polygamy will not significantly change the results.

The parameters relating to the genetic effects produced by various crosses will be described for each mechanism evaluated. In addition, examples will be given on how such calculations are made.

## TREND OF AN UNCONTROLLED POPULATION

To have a basis for evaluating the theoretical effects to be expected from any population suppression method, the trend of an uncontrolled population must be established. The species of pest is not necessarily relevant in this study, although reference to kinds of insects in which various genetic mechanisms have been observed will be mentioned from time to time. The dynamics of various kinds of insects vary depending on the species, the nature of the environment in which it exists, the density of the population, and the intensity of the density-dependent suppression forces. The rate of increase for a population may vary from less than onefold to many folds. However, for modeling purposes an average increase rate of fivefold per generation has been adopted as a realistic rate of increase for low populations of many insect species.

On the basis of these parameters an uncontrolled population would grow as shown below:

| *Generation* | *Number of insects* | |
|---|---|---|
| | ♀ | ♂ |
| Parent | 1,000 | 1,000 |
| $F_1$ | 5,000 | 5,000 |
| $F_2$ | 25,000 | 25,000 |
| $F_3$ | 125,000 | 125,000 |

### Trend of Populations Subjected to the Release of Male Insects That are Sterile Due to Radiation, Chemosterilants, Hybrid Sterilization, Cytoplasmic Incompatibility, and Compound Chromosomes

The released insects are assumed to be fully sterile, fully competitive in mating, and distributed so as to be accessible to all members of native populations. A total of 9,000 males are re-

leased during the first generation in one case and for each of the first three generations in the other. This will result in an initial overflooding ratio of 9 sterile to 1 fertile male competing for mates with the 1,000 females present. The results are shown in tables 1 and 2.

Calculations applicable for the two tables are as follows: *Fertile matings.*—Total fertile (normal) females in the population × the total fertile (normal) males ÷ by the total of all males = number of fertile matings. Number of fertile matings × 10 = the number of progeny, half of which are females and half males.

Example: *Fertile matings.*—1,000 F ♀ $\times \frac{1{,}000 \text{ F } ♂}{10{,}000 \text{ total}}$ = 100 matings × 10 = 500 ♀ + 500 ♂ progeny.

*Sterile matings.*—1,000 F ♀ $\times \frac{9{,}000 \text{ S } ♂}{10{,}000 \text{ total}}$ = 900 sterile matings = no progeny.

TABLE 1.—*Trend of an insect population subject to sterile male releases during 1 generation only*

| Generation | Normal, fertile insects | | Released males (sterile) | Ratio | Fertile matings | Progeny number | |
|---|---|---|---|---|---|---|---|
| | ♀ | ♂ | *Number* | *S:F* | *Number* | ♀ | ♂ |
| 1 (parent) | 1,000 | 1,000 | 9,000 | 9:1 | 100 | 500 | 500 |
| 2 ($F_1$) | 500 | 500 | 0 | | 500 | 2,500 | 2,500 |
| 3 ($F_2$) | 2,500 | 2,500 | 0 | | 2,500 | 12,500 | 12,500 |
| 4 ($F_3$) | 12,500 | 12,500 | 0 | | 12,500 | 62,500 | 62,500 |

TABLE 2.—*Trend of an insect population subjection to sterile male releases in 3 successive generations*

| Generation | Normal, fertile insects | | Released males (sterile) | Ratio | Fertile matings | Progeny number | |
|---|---|---|---|---|---|---|---|
| | ♀ | ♂ | *Number* | *S:F* | *Number* | ♀ | ♂ |
| 1 (parent) | 1,000 | 1,000 | 9,000 | 9:1 | 100 | 500 | 500 |
| 2 ($F_1$) | 500 | 500 | 9,000 | 18:1 | 26 3 | 131 | 131 |
| 3 ($F_2$) | 131 | 131 | 9,000 | 68 7:1 | 1 9 | 9 | 9 |
| 4 ($F_3$) | 9 | 9 | 0 | | 9 | 45 | 45 |

The models showing the theoretical trend of populations subjected to sterile-male releases have been depicted in many previous publications. However, they are again duplicated here to serve as a standard of comparison with results obtained from all other genetic mechanisms that will be considered in this study. Releases of sterile males for one generation only merely delays the growth of a population. Theoretically, the population in the different generation after the first will be only one-tenth as large as that of the uncontrolled population.

The population subjected to sterile-male releases for three successive generations, theoretically, will be reduced to near the point of extinction. However, if releases are discontinued, the nine insects of each sex in the $F_1$ generation will increase at the normal fivefold-increase rate. The releases of each successive generation have a progressively greater suppression impact because of the increasing ratio of sterile to fertile insects in the population. This outstanding characteristic of the genetic suppression procedure is well recognized and needs no further discussion.

The theoretical results, however, would be the same if sterile females were also released along with sterile males. However, only sterile-male releases are considered here because some of the sterility mechanisms, such as hybrid sterility or cytoplasmic incompatibility, may not cause sterility in the females.

From the standpoint of inherent efficiency, the suppression effect will be the same regardless of the way that males are sterilized. This does not mean, however, that in actual practice the results will be the same. Insects sterilized by radiation or chemicals may not be fully competitive in mating. Moreover, sperm from sterilized males may not be fully competitive with normal sperm, which would be detrimental when females are polygamous. Insects that are sterile because of hybridization, whether due to chromosome rearrangement, cytoplasmic incompatibility, or other genetic effects, may be more competitive in mating. On the other hand, male sterility produced by crossing species or races could produce hybrids that will not compete fully with males of the target species because of difference in behavior. Thus, one cannot predict which method of sterilization will be the most effective until careful field studies are made. However, for this comparative study we assume equal competitiveness in all cases, and conclude that the potential suppression of reproduction in a target pest population by releasing sterilized males will be the same regardless of the manner of male sterilization.

The theoretical effects due to sterile-male releases will be regarded as the standard of comparison for other genetic suppression mechanism to be considered in later evaluations.

## Trend of Population Subjected to the Release of Partially-Sterile Males (Inherited Sterility)

Several investigators have shown that certain insects (principally among Lepidoptera) receiving a given dosage of radiation or chemosterilants may be only partly sterilized, but the progeny of treated male parents × normal females are more sterile than the male parent. This has been called inherited sterility. When the theoretical effects of such sterility mechanisms were first calculated by the modeling procedure followed in this study, such action was shown to offer a potentially more effective suppression mechanism than the release of completely sterile males (*21*). This was true, even disregarding the probability that male parents receiving the lower sterilizing dosage will be more competitive in mating and produce more competitive sperm than fully sterile males.

It seems desirable to compare the potential of this mechanism of suppression with various other suppression mechanisms to be considered. As before, the theoretical effects will be calculated when releases are made for one generation only and for three successive generations. Treated males are assumed to be 60 percent sterile when mated to normal females, but both sexes of the $F_1$ progeny that are produced are regarded as 100 percent sterile when mated to normal insects or when they mate with each other. The results are shown in table 3.

Example of calculations (generation 2, for populations receiving three releases):

Fertile matings:

$$500\ F♀ \times \frac{500\ F♀}{11{,}300\ \text{total}} = 22.1\ F♀ \times 10$$
$$= 111\ F♀ + 111\ F♂\ \text{progeny.}$$

Partially sterile matings:[1]

$$500\ F♀ \times \frac{9{,}000\ 60\%\ S♂}{11{,}300\ \text{total}} = 398.2\ F♀ \times 60\%\ S♂ \times 4$$
$$= 796\ F♀ + 796\ F♂\ \text{progeny.}$$

[1] If the males are 60 percent sterile, the number of progeny from each mating will be 4, instead of 10 for normal matings.

TABLE 3.—*Trend of an insect population subjected to the release of partly sterile males during 1 and 3 generations*[1]

| Generation | Normal insects (fertile) | | Number and type of competing insects | | | Matings that produce progeny | Number and type of progeny | |
|---|---|---|---|---|---|---|---|---|
| | | | | Other | | | | |
| | ♀ | ♂ | Released | ♀ | ♂ | | ♀ | ♂ |
| | | | *Releases during generation 1* | | | | | |
| 1 (parent) | 1,000 | 1,000 | 9,000, 60% S | 0 | 0 | 100 F ♀ × F ♂ | 500 F | 500 F |
| | | | | | | 900 F ♀ × 60% S ♂ | 1,800 S | 1,800 S |
| 2 ($F_1$) | 500 | 500 | 0 | 1,800 S | 1,800 S | 108.7 F ♀ × F ♂ | 543 F | 543 F |
| 3 ($F_2$) | 543 | 543 | 0 | 0 | 0 | 543 F ♀ × F ♂ | 2,715 F | 2,715 F |
| 4 ($F_3$) | 2,715 | 2,715 | 0 | 0 | 0 | 2,715 F ♀ × F ♂ | 13,575 F | 13,575 F |
| | | | *Releases during generations 1, 2, and 3* | | | | | |
| 1 (parent) | 1,000 | 1,000 | 9,000, 60% S | 0 | 0 | 100 F ♀ × F ♂ | 500 F | 500 F |
| | | | | | | 900 F ♀ × 60% S ♂ | 1,800 S | 1,800 S |
| 2 ($F_1$) | 500 | 500 | 9,000, 60% S | 1,800 S | 1,800 S | 22.1 F ♀ × F ♂ | 111 F | 111 F |
| | | | | | | 398.2 F ♀ × 60% S ♂ | 796 S | 796 S |
| 3 ($F_2$) | 111 | 111 | 9,000, 60% S | 796 S | 796 S | 1.24 F ♀ × F ♂ | 6 F | 6 F |
| | | | | | | 100.8 F ♀ × 60% S ♂ | 202 S | 202 S |
| 4 ($F_3$) | 6 | 6 | 0 | 202 S | 202 S | <1 F mating | 0 | 0 |
| | | | | | | 208 S matings | 0 | 0 |

[1] F = fertile; S = sterile; 60% S = 60 percent sterile when mated to normal females.

Completely sterile matings:

$$500\ F\,♀ \times \frac{1{,}800\ S\,♂}{11{,}300\ \text{total}} = 80\ F\,♀ \times S\,♂ = \text{no progeny}$$

$$1{,}800\ S\,♀ \times \frac{500\ F\,♀}{11{,}300\ \text{total}} = 80\ S\,♀ \times F\,♂ = \text{no progeny}$$

$$1{,}800\ S\,♀ \times \frac{1{,}800\ S\,♂}{11{,}300\ \text{total}} = 287\ S\,♀ \times S\,♂ = \text{no progeny}$$

$$1{,}800\ S\,♀ \times \frac{9{,}000\ 60\%\ S\,♂}{11{,}300\ \text{total}} = 1{,}433\ S\,♀ \times 60\%\ ♂ = \text{no progeny.}$$

The release of partially sterile males having the effects projected is a substantially more effective suppression mechanism than the release of completely sterile males. This is indicated for releases for one generation or for three successive generations. Inherited sterility effects of the nature projected seem limited to species with holokinetic chromosomes, such as Lepidoptera and Hemiptera.

When releases are made in the first generation only, the sterile progeny present in the second generation ($F_1$), which are produced from matings by a partially sterile male and normal female parents, have a delayed suppression effect that cannot occur when completely sterile males are released. The population by the $F_2$ generation will number 2,715 fertile insects of each sex as compared with 12,500 of each sex when completely sterile males are released for one generation only. More than three times as many sterile males must be released in the first generation to achieve the theoretical suppression resulting from the release of partially sterile males for one generation. This does not take into account possible reduced competitiveness of males receiving the complete sterilizing dosages.

Whether the partially sterile males will, in fact, result in the degree of sterility shown during the $F_1$ generation remains to be seen. Investigations by various authors indicate that progeny from partially sterile males are affected in various ways. Sex-ratio distortion occurs in favor of males, and indications are that $F_1$ males from such crosses may not be fully competitive in sperm transfer. Both $F_1$ males and females show a low level of fertility when mated to normal insects. On the other hand, some persisting sterility in $F_2$ progeny that may be produced is evidenced.

Thus, the assumption of complete sterility for the progeny of a partially sterile male parent seems reasonable. Klassen and Creech (*18, pp 65-70*) have also calculated the effects of the release of partially sterile pink bollworm males based on genetic effects in the $F_1$ and $F_2$ generations, as observed by Graham and others (*13*).

Toba and others (*40*) conducted large cage tests comparing the suppression effects due to fully sterile males and partially sterile males of the cabbage looper, *Trichoplusia ni*. Greater suppression was achieved from the releases of partially sterile males. Thus, it seems that the conclusions reached in the theoretical appraisal are valid in principle, if not in exact detail.

The advantage of using partially sterile males over completely sterile males is also indicated when releases are made for three successive generations (table 3). When partially sterile male releases are made for three successive generations, the $F_3$ population, according to the parameters, would consist of six normal males and six normal females, but 202 sterile insects of each sex would also be present. This ratio of sterile to normal insects would lead to theoretical elimination of the population without making any additional releases. In the population subjected to the release of completely sterile males for three successive generations, nine fertile insects of each sex would remain in generation 4. Therefore, sterile male releases would have to continue in the fourth ($F_4$) generation to achieve theoretical elimination. Thus, on the basis of this analysis, sterile male releases would have to be made for four generations to have the same impact as releases for three generations when partially sterile males that transmit genetic effects to progeny are released.

We conclude that the inherited sterility mechanism resulting from the release of partially sterile males is potentially a more effective method of suppression than the sterile male release method. In addition, there may be further and more important advantages because of a higher degree of competitiveness of the males receiving the lower sterilization dosage.

In the application of the sterile-insect technique, the insects to be released are generally irradiated as pupae in the late stages of development or as adults soon after eclosion. The treatment of insects in the immature stages using moderate to high dosages generally causes severe damage and leads to death before adult emergence, or if there are survivors, they are likely to be deformed.

Recent investigations by Nielsen and Lambremont (unpub-

lished)[4] have shown that dosages of irradiation as low as 1-3 Krad when applied to immature stages of the greater wax moth, *Galleria mellonella* (Linnaeus), may cause no apparent adverse effects to eggs, larvae, or pupae but result in varying degrees of disruption of normal reproduction by the adults. Some earlier observations on the effects of low dosages of irradiation on survival and reproduction of emerging adults when eggs and larvae are treated were reported by Bartlett and others (*1*) on the pink bollworm (*Pectinophora gossypiella* (Saunders), and by Walker and others (*46, pp. 518-524*) on the sugarcane borer, *Diatraea saccharalis* (Fab.). However, these investigations appeared to have been too limited to indicate the potential usefulness of moths reared from irradiated immature stages. The effects of a range of dosages of irradiation applied to eggs, larvae, and pupae in different stages of development were investigated by Nielsen and Lambremont through the $F_1$ generation. The irradiation of immature stages with low dosages tended to affect the fecundity, fertility, and sex ratio of adults emerging from the untreated immature stages, and if suppression occurs with untreated insects, the $F_1$ progeny may inherit damage that causes partial sterility, sex distortion, or other adverse effects.

While some promising results were obtained from low dosages of irradiation applied to eggs, larvae, and pupae, the results obtained by irradiating 4- to 5-day-old eggs seemed to show most promise for the application of the inherited sterility principle. Inhibition of reproduction results from several causes. More males than females survive to the adult stage. When males or females mate with normal moths, egg hatch may be reduced. The survival rate of those that do hatch may be low, and again, males are more likely to survive than females. The $F_1$ progeny inherit sterility effects that cause a reduction in $F_2$ progeny in relation to normal insects. The total accumulative effect of such treatment applied to eggs seems to be much greater than that which can be achieved by the release of males that are completely sterile or by the release of males partially sterilized during the pupal or adult stage.

If the reported results with the greater wax moth can be substantiated and if similar effects are produced in other economic Lepidoptera, this mechanism may prove to be more effective and more economical than the conventional sterility technique.

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[4] Nielsen, R. A., ARS, U.S. Department of Agriculture, and Lambremont, E. N., Louisiana State University, "Radiation Biology of the Greater Wax Moth." [Unpublished.]

## Trend of Populations Subjected to the Release of "Strong-Race" Males

A genetic mechanism involving cross matings between males of a Japanese strain of the gypsy moth, *Porthetria dispar*, and females of the American strain of this pest was reported by Goldschmidt (*11, 12*). Males of the "strong" northern Japanese strain, according to Goldschmidt, when crossed with females of weak strains, such as those in Europe and the United States, resulted in female progeny that are sterile and die. The hybrid male progeny are fertile and carry the suppression factor designated as the strong-race factor. When these hybrid males are backcrossed with females of the regular American strain, half the females are sterile and die, and half are apparently normal. Half the males are normal weak race, and half carry the strong-race factor but are fertile. Downes (*7*) suggested that this genetic mechanism might be useful for the suppression of gypsy moth populations. While the genetic effects of various crosses and backcrosses have not been fully elucidated, we will assume that matings involving strong-race males to native weak-race females will result in the following type of lethal effects.

We will categorize various genotypes with the following symbols:

SR ♂ =strong-race males (homozygotes)=fertile
SR-WR ♂ =heterozygous strong-race, weak-race males =fertile
SR-WR ♀ =heterozygous females (lethal)
WR ♂ or WR ♀ =normal males or normal females=fertile

The various mating crosses in a natural population after SR males are released would produce the following types of progeny:

WR ♀ × SR ♂ = ½ SR-WR ♀ (die); ½ SR-WR ♂, fertile
WR ♀ × SR-WR ♂ = ¼ SR-WR ♀ (die); ¼ WR ♀, normal; ¼ SR-WR ♂, fertile; ¼ WR ♀, fertile
WR ♀ × WR ♂ =normal fertile males and females (WR)

The lethal effect of the strong-race factor can be transmitted only by males carrying the factor either in the homozygous or heterozygous state.

We assume, as before, that all males in the population are equally competitive in mating. The strong-race males will be released in the first generation only in one case and for three successive generations in the other. The natural population consists

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of 1,000 normal males and 1,000 normal females and will have a fivefold increase potential. Releases of males will be at a level of 9,000 each generation when releases are programed. The types of matings and progeny resulting from the releases are shown in table 4.

Example of calculations (generation 2, for population receiving three releases):

Normal fertile matings:

$$500\ \text{WR}\,♀ \times \frac{500\ \text{WR}\,♂}{14{,}000\ \text{total}} = 17.8\ \text{WR}\,♀ \times \text{WR}\,♂ \times 10$$
$$= 89\ \text{WR}\,♀ + 89\ \text{WR}\,♂.$$

Heterozygous WR male matings:

$$500\ \text{WR}\,♀ \times \frac{4{,}500\ \text{SR-WR}\,♂}{14{,}000\ \text{total}} = 160.7\ \text{WR}\,♀ \times \text{SR-WR}\,♂ \times 10$$
$$= 402\ \text{WR}\,♀ + 402\ \text{WR}\,♂$$
$$+ 402\ \text{SR-WR}\,♀\ (\text{die}) + 402\ \text{SR-WR}\,♂.$$

Homozygous SR male matings:

$$500\ \text{WR}\,♀ \times \frac{9{,}000\ \text{SR}\,♂}{14{,}000\ \text{total}} = 321.4\ \text{WR}\,♀ \times \text{SR}\,♂$$
$$= 1{,}607\ \text{SR-WR}\,♀\ (\text{die}) + 1{,}607\ \text{SR-WR}\,♂.$$

Total normal fertile WR progeny = 491 ♀ + 491 ♂.
Total heterozygous SR-WR = 2,009 ♂.

The calculations for the release of strong-race males for one generation show that this system would be substantially more effective than one generation release of sterile males of the regular strain. By the $F_1$ generation the population would consist of 5,328 normal WR females and 5,328 normal WR males. In addition, 1,547 heterozygous males would be carrying the SR-WR factors. The presence of these would exert some suppression in the next ($F_1$) generation, although this would not be highly significant because of the relatively low numbers present. The $F_1$ generation would consist of 23,642 normal insects (WR) of each sex. In contrast, when releases of fully sterile males of the regular strain are made, we would expect the population to build up to 62,500 of each sex by the $F_1$ generation.

The results of calculations to determine the effect of releases of strong-race males for three successive generations gave results that were not anticipated. We expected that the release of strong-race males for three successive generations would also show a greater suppression than the release of sterile males for three

successive generations. However, this is not true. As shown in table 2, the population subjected to sterile-male releases for three successive generations will be reduced to nine females and nine males in the $F_4$ generation. These figures will increase to 45 of each sex in the $F_1$ generation if sterile-male releases are not made in the $F_4$ generation. In the strong-race release system, the $F_4$ generation would consist of 319 normal weak-race males and 319 normal weak-race females and 2,135 males carrying the SR-WR factor. Without additional releases of males, the population in the $F_1$ generation will be 554 normal individuals of each sex and 347 males carrying the SR-WR factor, as compared with 45 normal males and 45 normal females when sterile-male releases are made for three generations.

These results are based on the assumption that all the males are fully competitive. It is possible that strong-race males, which would not have to be treated for sterilization, would be more vigorous and competitive than the regular weak-race males exposed to radiation or chemosterilants. On the other hand, there could be behavioral differences in the strong-race males that would reduce the effectiveness of the strong-race males below that calculated. Thus, we can only predict what impact the two methods would have on the assumption that the released males are equally competitive in all respects.

The importance of establishing appropriate models to calculate the theoretical effects of various potential genetic mechanisms is strongly demonstrated by this case. In an initial theoretical appraisal (unpublished) of the relative efficiency of strong-race and sterilized males of the regular strain, a comparison was made on the basis of releases for one generation only. This led to the conclusion that the strong-race males offer a significant advantage over sterilized males of the regular strain on the basis of equal competitiveness. We must modify our conclusion based on these studies and question if the genetic suppression mechanisms exhibited by the strong-race strain would, in fact, be an advantage over sterilization for the elimination of incipient gypsy moth populations. No calculations have been made to compare the relative efficiency of the two types of males when the release ratios are different from those programed in these models.

## Trends of an Insect Population Subjected to Releases of "Male-Producing" Strains of Insects

Certain genetic factors can lead to the production of progeny that are predominantly or all males. Craig and others (*3*) dis-

TABLE 4.—*The effect of strong-race (SR) males of the gypsy moth on population trends following releases against 1 and 3 generations*[1]

| Generation | Normal insects (fertile) | | Competing insects | | | Matings that produce progeny | Number and type of progeny | |
|---|---|---|---|---|---|---|---|---|
| | ♀ | ♂ | Released, ♂ | Other ♀ | Other ♂ | | ♀ | ♂ |
| | | | | *Releases during generation 1* | | | | |
| 1 (parent) | 1,000 WR | 1,000 | 9,000 SR | 0 | 0 | 100 WR♀ × WR♂ | 500 WR | 500 WR |
| | | | | | | 900 WR♀ × SR♂ | 4,500 SR WR (die) | 4,500 SR WR |
| 2 ($F_1$) | 500 WR | 500 WR | 0 | 0 | 4,500 SR WR | 50 WR♀ × WR♂ | 250 WR | 250 WR |
| | | | | | | 450 WR♀ × SR WR♂ | 1,125 WR | 1,125 WR |
| | | | | | | | 1,125 SR WR (die) | 1,125 SR WR |
| 3 ($F_2$) | 1,375 WR | 1,375 WR | 0 | 0 | 1,125 SR WR | 756 2 WR♀ × WR♂ | 3,781 WR | 3,781 WR |
| | | | | | | 619 NF♀ × SR WR♂ | 1,547 WR | 1,547 WR |
| | | | | | | | 1,547 SR WR (die) | 1,547 SR WR |
| 4 ($F_3$) | 5,328 WR | 5,328 WR | 0 | 0 | 1,547 SR WR | 4,129 WR♀ × WR♂ | 20,645 WR | 20,645 WR |
| | | | | | | 1,199 WR♀ × SR WR♂ | 2,997 WR | 2,997 WR |
| | | | | | | | 2,997 SR WR (die) | 2,997 SR WR |
| 5 ($F_4$) | 23,642 WR | 23,642 WR | 0 | 0 | 2,997 SR WR | | | |

| | | | | | | *Releases during generations 1, 2, and 3* | | |
|---|---|---|---|---|---|---|---|---|
| 1 (parent) | 1,000 WR | 1,000 WR | 9,000 SR | 0 | | 100 WR♀ ×WR♂ | 500 WR | 500 WR |
| | | | | | | 900 WR♀ ×SR♂ | 4,500 SR WR (die) | 4,500 SR WR |
| 2 ($F_1$) | 500 WR | 500 WR | 9,000 SR | 0 | 4,500 SR WR | 17 8 WR♀ ×WR♂ | 89 WR | 89 WR |
| | | | | | | 160 7 WR♀ ×SR WR♂ | 402 WR | 402 WR |
| | | | | | | | 402 SR WR (die) | 402 SR WR |
| | | | | | | 321 4 WR♀ ×SR♂ | 1,607 SR WR (die) | 1,607 SR WR |
| 3 ($F_2$) | 491 WR | 491 WR | 9,000 SR | 0 | 2,009 SR WR | 21 WR♀ ×WR♂ | 105 WR | 105 WR |
| | | | | | | 85 8 WR♀ ×SR WR♂ | 214 WR | 214 WR |
| | | | | | | | 214 SR WR (die) | 214 SR WR |
| | | | | | | 384 3 WR♀ ×SR♂ | 1,921 SR WR (die) | 1,921 SR WR |
| 4 ($F_3$) | 319 WR | 319 WR | 0 | 0 | 2,135 SR WR | 41 5 WR♀ ×WR♂ | 207 WR | 207 WR |
| | | | | | | 2,775 WR♀ ×SR WR♂ | 347 WR | 347 WR |
| | | | | | | | 347 SR WR (die) | 347 SR WR |
| 5 ($F_4$) | 554 WR | 554 WR | 0 | 0 | 347 SR WR | | | |

[1] WR = normal weak-race females and males, fertile; SR = strong-race homozygous males, fertile; SR WR = strong-race heterozygous males, fertile; SR-WR = heterozygous females, die.

covered strains of *Aedes aegypti* that produce up to 95 percent male progeny, and similar strains are known in *Drosophila*. Also, Wagoner (*43*) produced a strain of house flies in which males carrying a male progeny factor will produce all male progeny when they mate with normal females. We assume that most of these genetic mechanisms would function as described below. The male-producing factor will be called the M factor. Males homozygous for the factor will be designated as MM. The heterozygous males will be designated as Mm. However, half the males XXMm are incapable of mating. The XyMm males are fertile, capable of mating, and capable of transmitting the M (male-producing) factor. When such MmXy males cross with the normal females mmXX, the progeny segregate as follows: one-fourth are normal females; one-fourth are normal males; one-fourth are XyMm fertile males; and one-fourth are XXMm males incapable of reproducing.

We will calculate the effect of the release of 9,000 male-producing (MM) males into a normal population consisting of 1,000 females and 1,000 males. The effects of the male-producing strain will be calculated for male releases in the parent generation only and when releases are made in three successive generations. The results are shown in table 5. All basic parameters are as previously described.

The genetic mechanism that results in male progeny only when male-producing (MM) males cross with normal females has the same impact on reproduction as the strong-race male factor. The mechanism differs by producing some males that cannot reproduce, as contrasted with the strong-race factor by producing some females that die.

Example of calculations (generation 2, releases for three generations):

Normal fertile matings:

$$500\ \text{NF}\,♀ \times \frac{500\ \text{NF}\,♂}{1{,}400\ \text{total}} = 17.8\ \text{NF}\,♀ \times \text{NF}\,♂ \times 10$$
$$= 89\ \text{NF}\,♀ + 89\ \text{NF}\,♂.$$

Heterozygous XyMm male matings:

$$500\ \text{NF}\,♀ \times \frac{4{,}500\ \text{XyMM}\,♂}{14{,}000\ \text{total}} = 160.7\ \text{NF}\,♀ \times \text{XyMM F}\,♂$$
$$= 402\ \text{NF}\,♀ + 402\ \text{NF}\,♂ + 402\ \text{XyMm}\,♂, \text{ fertile},$$
$$+ 402\ \text{XXMm}\,♂, \text{ nonreproducing}.$$

Homozygous MM male matings:

$$500\ \text{NF}\,♀ \times \frac{9{,}000\ \text{MM}\,♂}{14{,}000\ \text{total}} = 321.4\ \text{NF}\,♀ \times \text{MM F}\,♂$$

$$= 1{,}607\ \text{XyMM F}\,♂$$

$$+ 1{,}607 \times \text{XXMm}\,♂, \text{ nonproducing}$$

Total normal fertile (NF) progeny—491 ♀ + 491 ♂.
Total heterozygous fertile males carrying the XyMm male-producing factor—2,009 ♂.

Releases of males for one generation only (parent) so as to overflood the natural male population by a factor of 9:1 cause greater suppression than the release of the same number of fully sterile males. Thus, on the basis of releases for one generation we conclude that the system is more effective than the sterile-male release technique. However, this does not hold when a comparison is made of releases for three successive generations. Like the release of strong-race males, the release of male-producing males for three successive generations is less effective than the sterile-male technique. Again this assumes equal competitiveness of males for both mechanisms. Greater competitiveness of males possessing the male-producing genetic factor or the strong-race factor, over males sterilized by radiation or by chemical means, could nullify the inherent advantage of the sterile-male technique. A possible advantage of the male-producing factor over the strong-race factor, if there is any, might be that the male-producing factor involves the use of strains of an insect that should be the same as the target strain from a behavioral standpoint. The strong-race mechanism involves the release of a different race of an insect which could have mating behavioral characteristics or ecological preferences that would differ from the target species in a natural environment.

## Trends of a Population Subjected to the Release of Insects That Inherit Sterility From Interspecific Crosses (Inherited Hybrid Male Sterility)

Laster (*26*) has recently reported on the occurrence of interspecific hybridization of *Heliothis virescens* (F) and *Heliothis subflexa* (Guenee). Information obtained in his studies may be briefly summarized as follows: When males of *H. virescens* are crossed with females of *H. subflexa* (which they are reported to

TABLE 5.—*The impact on reproduction when male-producing (MM) males are released against 1 and 3 generations*[1]

| Generation | Normal insects (fertile) | | Competing insects | | | Matings that produce progeny | Number and type of progeny | |
|---|---|---|---|---|---|---|---|---|
| | | | | Other | | | | |
| | ♀ | ♂ | Released, ♂ | ♀ | ♂ | | ♀ | ♂ |
| | | | | *Releases during generation 1* | | | | |
| 1 (parent) | 1,000 | 1,000 | 9,000 MM | 0 | 0 | 100 N♀ × N♂ | 500 NF | 500 NF |
| | | | | | | 900 N♀ × 900 MM♂ | 0 | 4,500 XyMm F |
| | | | | | | | | 4,500 XXMm, nonreproducing. |
| 2 ($F_1$) | 500 | 500 | 0 | 0 | 4,500 XyMm F | 50 N♀ × 50 N♂ | 250 NF | 250 NF |
| | | | | | | 450 N♀ × XyMm F♂ | 1,125 NF | 1,125 NF |
| | | | | | | | | 1,125 XyMm F |
| | | | | | | | | 1,125 XXMm, nonreproducing. |
| 3 ($F_2$) | 1,375 | 1,375 | 0 | 0 | 1,125 XyMm F | 756 2 N♀ × N♂ | 3,781 NF | 3,781 NF |
| | | | | | | 619 N♀ × XyMm F♂ | 1,547 NF | 1,547 NF |
| | | | | | | | | 1,547 XyMm F |
| | | | | | | | | 1,547 XXMm, nonreproducing |
| 4 ($F_3$) | 5,328 | 5,328 | 0 | 0 | 1,547 XyMm F | 4,129 1 N♀ × N♂ | 20,654 NF | 20,654 NF |
| | | | | | | 1,199 N♀ × XyMm F♂ | 2,997 NF | 2,997 NF |
| | | | | | | | | 2,997 XyMm F |
| | | | | | | | | 2,997 XXmM, |
| | | | | | | | | 2,997 XXMm, nonreproducing. |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 5 ($F_4$) | 23,644 | 23,644 | 0 | 0 | 2,997 XyMm F | | | |
| *Releases during 3 generations* | | | | | | | | |
| 1 (parent) | 1,000 | 1,000 | 9,000 MM | 0 | 0 | 100 N♀ × N♂ | 500 NF | 500 NF |
| | | | | | | 900 N♀ × 900 MM♂ F | 0 | 4,500 XyMm F |
| | | | | | | | | 4,500 XXMm, nonreproducing. |
| 2 ($F_1$) | 500 | 500 | 9,000 | 0 | 4,500 XyMm F | 17.8 N♀ × N♂ | 89 NF | 89 NF |
| | | | | | | 160.7 N♀ × XyMm♂ F | 402 NF | 402 NF |
| | | | | | | | | 402 XyMm F |
| | | | | | | | | 402 XXMm, nonreproducing. |
| | | | | | | 321.4 N♀ × MM♂ F | 0 | 1,607 XyMm F |
| | | | | | | | | 1,607 XXMm, nonreproducing. |
| 3 ($F_2$) | 491 | 491 | 9,000 MM | 0 | 2,009 XyMm F | 21 N♀ × N♂ | 105 NF | 105 NF |
| | | | | | | 85.8 N♀ × XyMm♂ F | 214 NF | 214 NF |
| | | | | | | | | 214 XyMm F |
| | | | | | | | | 214 XXMm, nonreproducing. |
| | | | | | | 384.3 N♀ × MM♂ F | 0 | 1,921 XyMm F |
| | | | | | | | | 1,921 XXMm, nonreproducing. |
| 4 ($F_3$) | 319 | 319 | 0 | 0 | 2,135 XyMm F | 41.5 N♀ × N♂ | 207 NF | 207 NF |
| | | | | | | 277.5 N♀ × XyMm♂ F | 347 NF | 347 XyMm F |
| | | | | | | | | 347 XXMm, nonreproducing. |
| 5 ($F_4$) | 554 | 554 | 0 | 0 | 347 XyMm F | | | |

NF = normal fertile males and females; M = homozygous for male-producing factor; XyMm = heterozygous for male-producing factor F = fertile; XXMm = heterozygous for male-producing factor, nonreproducing.

do readily in the laboratory), the male progeny are completely sterile when mated to *H. virescens* or *H. subflexa* females. The $F_1$ hybrid males are also sterile when they cross with the $F_1$ hybrid females. However, substantial reproduction occurs when the $F_1$ hybrid females are backcrossed with *H. virescens* males. The $F_1$ hybrid females when crossed with *H. subflexa* males produced no progeny. On the basis of these effects, Laster proposed that the hybrid male progeny from the *H. virescens* ♂ × *H. subflexa* ♀ matings may be useful in the application of the sterile-male technique. A differential in size and time for development of hybrid male and hybrid female pupae would facilitate the separation of sexes. Further studies by Proshold and LaChance indicate that the sterility of male progeny is caused by their inability to transfer eupyrene sperm, and that this inability may be caused by difficulties in pairing of the chromosomes during meiosis.[5]

The findings of Laster are, indeed, interesting. As he points out, the male hybrids may provide a superior sterile male for suppressing *H. virescens*, one of the Nation's most damaging pests. However, based on the releases of males only, the effects should be the same as those produced by fully sterile and competitive males. The theoretical effects of releasing both hybrid sterile males and hybrid sterile females were calculated. The results were surprising and spectacular. Since the $F_1$ hybrid females are fertile when backcrossed to *H. virescens* males, one is likely to assume that the release of the fertile hybrid females along with the sterile hybrid males would be a disadvantage in suppression and would create risks from the standpoint of crop damage. We will show, however, that this is not true if the genetic effects of the various crosses are as reported by Laster.

According to data presented by Laster, the sterility persists in the $F_2$ hybrid males ($F_1$ hybrid female backcrossed to *H. virescens* males) and fertility also persists in the $F_2$ hybrid females. Indeed, these effects are observed for eight recurrent backcrosses.[6] On the basis of these observations, the effects on reproduction of the various crosses can be summarized as follows:

[5] LeChance, L. E., Personal communication, Metabolism and Radiation Research Laboratory, Fargo, N. Dak. 58102.

[6] Laster, M. L., Personal communication, Delta Branch, Mississippi State Agricultural Experiment Station, Stoneville, Miss. 38776.

Parent: *H. virescens* ♂ × *H. subflexa* ♀ = $F_1$ hybrid males and females.

$F_1$ hybrids:

- $F_1$ hybrid ♂ × *H. virescens* ♀ = no pregeny
- $F_1$ hybrid ♂ × *H. subflexa* ♀ = no progeny
- $F_1$ hybrid ♂ × $F_1$ hybrid ♀ = no progeny
- $F_1$ hybrid ♀ × *H. subflexa* ♂ = no progeny
- $F_1$ hybrid ♀ × *H. virescens* ♂ = reproduction with viable $F_2$ progeny.

$F_2$ hybrids:

- $F_2$ hybrid ♂ × *H. virescens* ♀ = no progeny
- $F_2$ hybrid ♂ × *H. subflexa* ♀ = no progeny
- $F_2$ hybrid ♂ × $F_2$ hybrid ♀ = no progeny
- $F_2$ hybrid ♀ × *H. subflexa* ♂ = no progeny
- $F_2$ hybrid ♀ × *H. virescens* ♂ = reproduction with viable progeny.

$F_3$ hybrids:

- $F_3$ hybrid ♂ × *H. virescens* ♀ = no progeny
- $F_3$ hybrid ♀ × *H. virescens* ♂ = reproduction with viable progeny.

Using the data presented, calculations were made to determine the effect of releases of both sexes of the hybrid progeny when released to compete in a normal *H. virescens* population. The release of the sterile hybrid males only theoretically would have the same effect as the release of males sterilized by radiation or by chemosterilants. The same basic parameters are used as described for calculating the effects of other genetic mechanisms. Calculations were made for releases made for one generation only and for two successive generations. (Theoretically, releases would not need to be programed for three generations, as considered for other genetic mechanisms.) The results of the calculations are presented in table 6.

The various insects will carry the following symbol designations:

- N = normal
- F = fertile
- S = sterile
- H = $F_1$ hybrids
- $H_1$ = $F_1$ hybrids (progeny from the backcross of $F_1$ hybrid ♀ to *H. virescens* ♂).

$H_1$ = $F_1$ hybrids (progeny from the backcross of $F_1$ hybrid ♀ to *H. virescens* ♂).
$H_{1+}$ = $F_1$ and F hybrids

Example of calculations (generation 2, when releases are made in generations 1 and 2):

Normal fertile matings:

$$\frac{500 \text{ F} \female \times 500 \text{ F} \male}{14{,}000 \text{ total}} \quad 17.9 \text{ matings} \times 10 \quad 89 \text{ N F} \female + 89 \text{ N F} \male .$$

Hybrid fertile females × normal males =

$$13{,}500 \text{ H F} \female \times \frac{500 \text{ F} \male}{14{,}000} \quad 482 \text{ matings} \times 10$$
$$= 2{,}411 \ H_{1+} \text{ F } 3{,}411 \ H_{1+} \text{S}.$$

Sterile matings:

$$500 \text{ F} \female \times \frac{13{,}000 \text{ S} \male}{14{,}000} \quad 482 \text{ matings} = \text{no progeny}$$

$$13{,}500 \text{ H F} \female \times \frac{13{,}500 \text{ S} \male}{14{,}000} \quad 13{,}018 \text{ matings} = \text{no progeny.}$$

Total matings 14,000.

The calculated effects due to the release of both hybrid sterile males and hybrid fertile females for one generation are, by far, the most impressive of the various genetic mechanisms considered thus far. Even though a large number of fertile hybrid females are released along with the sterile hybrid males, this does not add to the reproductive capability of the population in comparison with an untreated population because of the nullifying effect of the sterile hybrid males. The second generation ($F_1$) population would total 10,000 including both sexes, which would be the same as the normal untreated population increasing at a fivefold rate. Of this number, however, 4,500 would be hybrid sterile males, and 500 would be fertile males competing to mate with the 5,000 fertile females. Thus, only 500 fertile matings would be expected, in contrast with 5,000 for an uncontrolled population. The population would theoretically be further reduced by half to a total of 5,000 in generation 3 ($F_2$), and only 250 fertile matings would be expected as compared with 25,000 expected fertile matings for an uncontrolled population. By generation 4 the number of fertile matings would be 125 as compared with 125,000 for an uncontrolled population.

TABLE 6.—*Impact of releases of sterile hybrid males and fertile hybrid females that transmit sterility to male progeny and the sterility factor to fertile female progeny*

| Generation | Normal insects fertile | | Competing insects | | | | Matings that produce progeny | Number and type of progeny | |
|---|---|---|---|---|---|---|---|---|---|
| | | | Released | | Other | | | | |
| | ♀ | ♂ | ♀ | ♂ | ♀ | ♂ | | ♀ | ♂ |
| *Releases during generation 1* | | | | | | | | | |
| 1 (parent) | 1,000 | 1,000 | 9,000 $H_1F$ | 9,000 $H_1S$ | 0 | 0 | 100 N ♀ × N ♂ | 500 N | 500 N |
| | | | | | | | 900 $H_1F$ ♀ × N ♂ | 4,500 $H_1F$ | 4,500 $H_1S$ |
| 2 ($F_1$) | 500 N | 500 N | 0 | 0 | 4,500 $H_1F$ | 4,500 $H_1S$ | 50 N ♀ × N ♂ | 250 N | 250 N |
| | | | | | | | 450 $H_1F$ ♀ × N ♂ | 2,250 $H_1F$ | 2,250 $H_1S$ |
| 3 ($F_2$) | 250 N | 250 N | 0 | 0 | 2,250 $H_1F$ | 2,250 $H_1S$ | 25 N ♀ × 25 N ♂ | 125 N | 125 N |
| | | | | | | | 225 $H_1F$ ♀ × N ♂ | 1,125 $H_1F$ | 1,125 $H_1S$ |
| 4 ($F_3$) | 125 N | 125 N | 0 | 0 | 1,125 $H_1F$ | 1,125 $H_1S$ | 12 5 N ♀ × N ♂ | 62 5 N | 62 5 N |
| | | | | | | | 112 5$H_1$ F ♀ × N ♂ | 562 5 $H_1F$ | 562 5 $H_1S$ |
| *Releases during generations 1 and 2* | | | | | | | | | |
| 1 (parent) | 1,000 | 1,000 | 9,000 $H_1F$ | 9,000 $H_1S$ | 0 | 0 | 100 N ♀ × N ♂ | 500 N F | 500 N F |
| | | | | | | | 900 $H_1F$ ♀ × N ♂ | 4,500 $H_1F$ | 4,500 $H_1S$ |
| 2 ($F_1$) | 500 | 500 | 9,000 $H_1F$ | 9,000 $H_1S$ | 4,500 $H_1F$ | 4,500 $H_1S$ | 17 9 N ♀ × N ♂ | 89 N F | 89 N F |
| | | | | | | | 482 $H_{1+1}F$ ♀ × N ♂ | 2,411 $H_{1+1}F$ | 2,411 $H_{1+1}S$ |
| 3 ($F_2$) | 89 | 89 | 0 | 0 | 2,411 $H_{1+1}F$ | 2,411 $H_{1+1}S$ | 3 2 N ♀ × N ♂ | 16 N F | 16 N F |
| | | | | | | | 85 8 $H_{1+1}F$ ♀ × N ♂ | 429 $H_{1+1}F$ | 429 $H_{1+1}S$ |
| 4 ($F_3$) | 16 | 16 | 0 | 0 | 429 $H_{1+1}F$ | 429 $H_{1+1}S$ | 6 N ♀ × N ♂ | 3 | 3 |
| | | | | | | | 15 4 $H_{1+1}F$ ♀ × N ♂ | 77 $H_{1+1}F$ | 77 $H_{1+1}S$ |

[1] The population, theoretically, would continue to decline by ½ so long as the genetic mechanism persists in the fertile hybrid females and is transmitted to produce sterility in male progeny.

In the sterile-male release system the number of fertile matings, as may be noted in table 1, would be reduced by 90 percent in generation 1, but the number of matings would increase by five-fold each generation thereafter. The potential advantage of the hybrid strain is thus fully apparent on the basis of releases for one generation only.

The potential advantage of the hybrid strain over the sterilized *H. virescens* males is equally, if not more impressive, when compared on the basis of successive releases. When two successive releases are made, the number of fertile matings in generation 2 would total 500. In generation 3 the number of fertile matings would be 89 and in generation 4, 16. However, by generation 4 the 16 matings would all involve normal males and hybrid fertile females, so no normal males would be produced. Theoretical elimination would occur in the fifth generation.

If the effects on reproduction of the hybrid moths as reported by Laster are confirmed through additional investigations, this could provide one of the most powerful genetic mechanisms known for the suppression of *H. virescens*. It is potentially more effective than the inherited sterility factor using partially sterilized moths or the genetic effects, which were considered earlier, resulting from the irradiation of eggs. Research is needed to determine the behavior and competitiveness of the sterile hybrid males and the hybrid females. Will the sterile hybrid males be competitive in matings and in sperm production? Will the hybrid fertile females be competitive with *H. virescens* females in the attraction of both normal males and hybrid sterile males? Will the hybrid males and females seek the same habitats in the ecosystem? Will the hybrid sterility effects persist, and will the behavior of the hybrids be more and more like *H. virescens* as the backcrosses continue? These questions need answers. However, on the basis of the reported genetic effects the calculations show a great potential for the inherited hybrid sterility mechanisms.

The value of the modeling procedure developed for appraising the efficiency of various genetic mechanisms is clearly indicated by the result of this appraisal of the theoretical effects of the genetic mechanism described. One might expect that the release of fertile hybrid females, along with sterile hybrid males, to increase the natural population and pose a risk of crop damage above that of the natural population. However, by employing simulated population models to calculate probable effects of both male and female releases, the unique suppression effect was clearly indicated.

## Trends of a Population Subjected to the Release of Both Sexes of a Compound Chromosome Strain

The genetic effects of compound chromosomes were previously described and the potential usefulness of the suppression mechanism, especially if a conditional lethal factor were linked with the compound chromosome, discussed.

To appraise the potential effect of the suppression mechanism, we will deviate somewhat from the format followed in earlier models. The natural pest population will consist of 1,000 of each sex as before. A nondiapausing strain bearing a compound chromosome will be released at the rate of 9,000 females and 9,000 males as before. However, releases are programed for one generation only.

The compound chromosome arrangement in the strain is assumed to result in 75 percent self-sterility when intramatings occur. All matings involving individuals bearing the compound chromosome and normal insects will produce no progeny because of chromosome imbalance. Normal matings will result in 10 adult progeny (fivefold increase). The matings involving individuals bearing the compound chromosome would also increase fivefold except for the self-sterile factor. Thus, with an increase potential of fivefold but a self-sterile factor of 75 percent (25 percent fertility), the actual increase will be slight. For example, if 100 insects are involved in reproduction, the expected progeny will be $0.25 \times 5 \times 100 = 125$. Thus, some increase in the number of compound chromosome-bearing insects will result from intermatings involving the compound chromosome strain. Also, the expected increase from normal $\times$ normal insects will occur. The total number of insects would, however, be much below that of an uncontrolled population.

The most important suppression factor would be the conditional nondiapause characteristic that would be expressed during the winter. All insects bearing the gene for inability to diapause would fail to survive. This conditional lethal gene is assumed to occur only on the compound chromosome.

On the basis of the parameters described, the population trend and final result by the fourth generation would be as calculated below.

$N \times N$ = matings involving normal males and females;

$N \times C$ and $C \times N$ = matings involving compound chromosome-bearing males and females;

C × C = matings between males and females which have a compound chromosome.

To illustrate the impact of releasing both sexes of a strain with compound chromosomes, consider a wild population of 1,000 males and 1,000 females that is overflooded once with 9,000 males and 9,000 females of a nondiapause strain with a compound chromosome. The results are shown in table 7.

The calculations show that by the fourth generation the population consists of no normal males, no normal females, 18,654 compound chromosome-bearing males, and 18,654 compound chromosome-bearing females. The expected total of 37,306 com-

TABLE 7.—*Impact of a single release of a compound chromosome-bearing strain with a conditional lethal, such as the inability to diapause on a native population*

| Cross and number of matings | Female progeny | Male progeny | Total |
|---|---|---|---|
| | *First generation* | | |
| N×N 100 | 500 N | 500 N | 1,000 N |
| N×C 900 (sterile) | 0 | 0 | 0 |
| C×N 900 (sterile) | 0 | 0 | 0 |
| C×C 8,100 (0.25 fertile) | 10,125 C | 10,125 C | 20,250 C |
| | *Second generation* | | |
| N×N 23.6 | 118 N | 118 N | 236 N |
| N×C sterile | 0 | 0 | 0 |
| C×N sterile | 0 | 0 | 0 |
| C×C 9,648 (0.25 fertile) | 12,060 C | 12,060 C | 24,120 C |
| | *Third generation* | | |
| N×N 1.4 | 6 N | 6 N | 12 N |
| N×C sterile | 0 | 0 | 0 |
| C×N sterile | 0 | 0 | 0 |
| C×C 11,944 (0.25 fertile) | 14,930 C | 14,930 C | 29,860 C |
| | *Fourth generation* | | |
| N×N 9 | 0 | 0 | 0 |
| N×C sterile | 0 | 0 | 0 |
| C×N sterile | 0 | 0 | 0 |
| C×C 14,923 (0.25 fertile) | 18,654 | 18,654 | 37,308 (lack ability to diapause) |

pound chromosome-bearing insects would carry the genes for inability to diapause and thus would be eliminated.

If such genetic mechanism would be developed in strains of insect pests that are good candidates for the autocidal control system, it should prove to be an efficient and powerful mechanism for population suppression. It would be comparable in efficiency to the inherited hybrid sterility mechanisms previously considered. Releases for only one generation during the permissive period should lead to extinction of the natural population. The self-sterility feature would slow down the rate of growth of the total population until the conditional lethal effect is expressed. The release of insects during a series of generations would not add to the effectiveness. In fact, according to the parameters, this would be detrimental and could lead to an economic population before the nondiapause or some other conditionally lethal factor is expressed.

## Trends of an Insect Population Subjected to Releases by Strains With a Meiotic-Drive Coupled to a Dominant Conditionally Lethal Gene

The search for meiotic-drive factors seems to have decreased somewhat with the demonstration that several such factors could not be fixed because of recessive sterility (*9*). However, there is no *a priori* reason to assume that all meiotic-drive factors are associated with recessive sterility.[1] For this appraisal we assume that a meiotic-drive factor can be fixed in the homozygous condition and tightly coupled to a dominant conditionally lethal gene. Such a gene might determine the inability to diapause, or some other trait that would prevent survival under certain conditions. The strain with the meiotic-drive factor coupled to the dominant conditional lethal gene is designated DD, and the normal strain is designated NN. Further, we assume that an ND hybrid of either sex would produce 10 percent N gametes and 90 percent D gametes. Therefore, the ratio of progeny for the cross NN $\times$ ND would be 0.1 NN:0.9 ND, and the ratio of progeny for the cross ND $\times$ ND would be 0.1 NN:0.18 ND:0.8100 DD. Thus, if a natural population of 1,000 NN ♀ and 1,000 NN ♂ was overflooded in each generation with 9,000 DD ♂, the various progeny for the first two generations would be derived as shown below. The number of adult progeny per mating is assumed to be 10.

[1] See ftnt. 2, p. 7.

| Generation | Mating | | Progeny | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | Female | | | Male | | |
| | | | NN | ND | DD | NN | ND | DD |
| 1 | NN♀ × NN♂ | 1,000 × 1,000/10,000 = 100 matings | 500 | | | 500 | | |
| | NN♀ × DD♂ | 1,000 × 9,000/10,000 = 900 matings | | 4,500 | | | 4,500 | |
| 2 | NN♀ × NN♂ | 500 × 500/14,000 = 17.0 matings | 90 | | | 90 | | |
| | NN♀ × ND♂ | 500 × 4,500/14,000 = 160.7 matings | 80 | | | 80 | | |
| | NN♀ × DD♂ | 500 × 9,000/14,000 = 321.4 matings | | 1,607 | | | 1,607 | |
| | ND♀ × NN♂ | 4,500 × 500/14,000 = 160.7 matings | 80 | | | 80 | | |
| | ND♀ × ND♂ | 4,500 × 4,500/14,000 = 1,446.4 matings | 72 | | | 72 | | |
| | ND♀ × DD♂ | 4,500 × 9,000/14,000 = 2,892.9 matings | | 1,446 | 13,018 | | 1,446 | 13,018 |

In this way we calculated the effect of the release of 9,000 meiotic-drive males (DD) into a normal population consisting of 1,000 females and 1,000 males during the parental generation only and during generations 1, 2, and 3 (table 8). The table also shows the results of releasing 9,000 meiotic-drive males and 9,000 meiotic-drive females into a normal population of 1,000 females and 1,000 males during the parent generation.

The results show that far greater suppression can be achieved with a meiotic drive coupled with a dominant conditional lethal gene than with releases of sterile males and most of the other genetic suppression systems, when releases are made for the parental generation only. However, the advantage of three successive releases of males only over a single release of males only is not particularly great. The release of both sexes of the hypothetical strain at the release ratio in this appraisal would have the greatest effect. A single release of both sexes of the meiotic-drive strain would have a greater effect than three successive releases of males only.

If a genetic mechanism as described should be available for a given pest, the released insects would provide no suppression until the conditionally lethal factor had been expressed. Therefore, the usual control method would be necessary if the population reached the economic threshold level before season's end. However, the release of males only should not influence the total number of progeny produced. If both sexes were released to produce the greatest final effect, the natural population would no doubt have to be greatly suppressed by conventional methods. Further, all or part of the population could be replaced first by a strain carrying the meiotic-drive factor and the coupled dominant conditionally lethal gene. In this way the releases would not create hazards above that of a normal population that had not been suppressed.

## Trends of Populations Subjected to Releases of Strains With Dominant or Recessive Conditionally Lethal Genes

Unlike most of the genetic mechanisms considered in this appraisal, conditional lethals do not provide any suppression until the onset of restrictive conditions. The release of males only with conditional lethals would not add more progeny to subsequent generations than would occur in the absence of releases. Nevertheless, the pest population would increase until the onset of restrictive conditions. To prevent such an increase to levels above

TABLE 8.—*Genotypic and population trends when a native population (NN) of 1,000 males and 1,000 females is overflooded with individuals (DD) homozygous for a meiotic-drive factor tightly coupled with a dominant conditionally lethal gene (DD). The rate of increase is fivefold, and the conditionally lethal gene would not be expressed until the 4th generation*

| Generation | Male progeny | | | Female progeny | | | Total |
|---|---|---|---|---|---|---|---|
| | NN | ND | DD | NN | ND | DD | |
| | | | *9,000 DD males released during generation 1* | | | | |
| 1 parent | 500 | 1,500 | | 500 | 4,500 | | 10,000 |
| 2 $F_1$ | 902 | 7,695 | 16,402 | 902 | 7,695 | 16,402 | 49,998 |
| 3 $F_2$ | 559 | 15,602 | 108,841 | 559 | 15,602 | 108,841 | 250,004 |
| 4 $F_3$ | [1] 180 | 20,835 | 604,007 | [1] 180 | 20,835 | 604,007 | 1,250,044 |
| | | | *9,000 DD males released during generations 1, 2, and 3* | | | | |
| 1 parent | 500 | 4,500 | | 500 | 4,500 | | 10,000 |
| 2 $F_1$ | 322 | 5,802 | 18,875 | 322 | 5,802 | 18,876 | 49,998 |
| 3 $F_2$ | 118 | 7,588 | 117,292 | 118 | 7,588 | 117,292 | 249,996 |
| 4 $F_3$ | [1] 30 | 8,735 | 614,377 | [1] 30 | 8,735 | 614,377 | 1,246,284 |
| | | | *9,000 DD males and 9,000 females released during generation 1* | | | | |
| 1 parent | 500 | 9,000 | 40,500 | 500 | 9,000 | 40,500 | 100,000 |
| 2 $F_1$ | 196 | 13,608 | 236,196 | 196 | 13,608 | 236,196 | 500,000 |
| 3 $F_2$ | 15 | 14,871 | 1,231,781 | 15 | 14,871 | 1,231,781 | 5,493,334 |
| 4 $F_3$ | [1] 9 | 14,874 | 6,217,690 | [1] 9 | 14,874 | 6,232,573 | 12,465,146 |

[1] Survivors.

the economic threshold, supplemental control measures must be applied until the effects of the genetic mechanism become effective. Therefore, we cannot directly compare the efficiency of conditional lethals into that portion of the wild population that suppression.

Two approaches to holding the population in check during permissive conditions are proposed: (1) Conventional measures and (2) inducing a sufficient level of sterility into the release strain to hold the pest population static while at the same time infusing conditional lethals into that portion of the wild population that is reproducing. This system was suggested by Klassen and others (*16*). We calculated the effects of overflooding only the parental generation of a population held static by conventional means, and of overflooding the parental, $F_1$, and $F_2$ generations with males

or males and females of a strain homozygous for a monofactorial conditional lethal trait (AA) (table 9).

One release of males only would provide a suppression of about 70 percent if the dominant conditionally lethal gene was expressed in the $F_2$, $F_3$, or $F_4$ generations. If both sexes were released, the suppression would be 99 percent if the gene was expressed in the $F_1$, $F_2$, $F_3$, or $F_4$ generation (table 9).

Three releases of males only would provide a suppression of 97 percent or more if the dominant conditionally lethal gene were expressed in the $F_3$ or $F_4$ generation and about 92 percent suppression if expression occurred in the $F_2$ generation. If both sexes were released, the suppression would be greater than 99 percent if the gene were expressed in the $F_2$ or subsequent generations (table 9).

If a static native population (aabb) of 1,000 males and 1,000 females were overflooded once with 9,000 males homozygous for two dominant conditionally lethal genes (AABB), the suppression

TABLE 9.—*Genotypic trends when a native population (aa) of 1,000 males and 1,000 females is overflooded once in the parental generation and in generations 1, 2, and 3 with insects homozygous for one dominant conditionally lethal gene (AA). In one model for each type of release 9,000 males only are released and in the other, 9,000 males and 9,000 females are released*[1]

| Genotype | Males only released | | | | Males and females released | | | |
|---|---|---|---|---|---|---|---|---|
| | $F_1$ | $F_2$ | $F_3$ | $F_4$ | $F_1$ | $F_2$ | $F_3$ | $F_4$ |
| | *Releases during generation 1 only* | | | | | | | |
| aa | 200 | 605 | 605 | 605 | 20 | 20 | 20 | 20 |
| Aa | 1,800 | 990 | 990 | 990 | 360 | 360 | 360 | 360 |
| AA | | 405 | 405 | 405 | 1,620 | 1,620 | 1,620 | 1,620 |
| Total | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 |
| | *Releases during generations 1, 2, and 3* | | | | | | | |
| aa | 200 | 61 | 33 | 155 | 20 | 0 | 0 | 0 |
| Aa | 1,800 | 1,089 | 1,049 | 804 | 360 | 43 | 5 | 5 |
| AA | | 850 | 918 | 1,040 | 1,620 | 1,957 | 1,995 | 1,995 |
| Total | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 |

[1] The population is held static by conventional means.

would be roughly 90 percent if the conditionally lethal trait were expressed in the $F_1$, $F_2$, $F_3$, or $F_4$ generations. (We assume that a single dominant allele would be fully penetrant.) On the other hand, if 9,000 males and 9,000 females had been released, the corresponding suppression would have been greater than 99 percent (table 10). If males only had been released for three consecutive generations, the suppression would have increased from 90 percent if the trait were expressed in the $F_1$, to more than 99 percent if it were expressed in the $F_4$ generation (table 11). If males and females were released for three consecutive generations, suppression would be complete if the conditionally lethal trait were expressed after the $F_2$ generation (table 11).

Thus the percentage of suppression in the $F_4$ generation obtained by releasing insects with one or two conditionally lethal genes or traits into a native population held static by conventional means is as follows:

| | *Dominant conditional lethals* | *Recessive conditional lethals* |
|---|---|---|
| 1 gene: | | |
| Males only, 1 release | 70 | 20 |
| Males only, 3 releases | 92 | 52 |
| Both sexes, 1 release | 99 | 81 |
| Both sexes, 3 releases | 100 | 100 |
| 2 genes: | | |
| Males only, 1 release | 91 | 36 |
| Males only, 3 releases | 100 | 86 |
| Both sexes, 1 release | 100 | 95 |
| Both sexes, 3 releases | 100 | 100 |

Clearly, dominant conditional lethals have the potential to suppress populations drastically. A monofactorial recessive conditional lethal will not provide strong suppression unless both sexes are released for several generations, and this is true, although to a lesser extent, even if the release strain has two recessive conditional lethals.

The use of partial sterility (that is, not inherited sterility) in the release strain rather than conventional methods for holding the pest population in check while one or two dominant conditionally lethal genes are being infused into the gene pool of the wild population was considered. The results of these computations are not shown here; however, they indicated that at a release ratio of only 9:1, the joint use of partial sterility and dominant

TABLE 10.—*Genotypic trends when a native population (aabb) of 1,000 males and 1,000 females is overflooded once in the parental generation with insects homozygous for 2 dominant conditionally lethal genes (AABB). In one model, 9,000 males only are released, and in the other 9,000 males and 9,000 females are released*[1]

| Genotype | Males only released, generation | | | | Males and females released, generation | | | |
|---|---|---|---|---|---|---|---|---|
| | $F_1$ | $F_2$ | $F_3$ | $F_4$ | $F_1$ | $F_2$ | $F_3$ | $F_4$ |
| aabb | 200 | 211 | 196 | 190 | 20 | 6 | 2 | 1 |
| Aabb | 0 | 293 | 296 | 298 | 0 | 10 | 9 | 7 |
| AAbb | 0 | 101 | 112 | 117 | 0 | 4 | 9 | 12 |
| aaBb | 0 | 293 | 296 | 298 | 0 | 10 | 9 | 7 |
| AaBb | 1,800 | 495 | 492 | 491 | 360 | 196 | 126 | 95 |
| AABb | 0 | 202 | 202 | 201 | 0 | 154 | 225 | 259 |
| aaBB | 0 | 101 | 112 | 117 | 0 | 4 | 9 | 12 |
| AaBB | 0 | 202 | 202 | 201 | 0 | 154 | 225 | 259 |
| AABB | 0 | 101 | 91 | 87 | 1,620 | 1,462 | 1,386 | 1,349 |
| Total | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 |

[1] The population is held static by conventional means.

conditionally lethal genes is less efficient than the use of complete interstrain sterility. At this ratio, also, the method is decidedly less efficient with, say, 90 percent sterility than with 95 percent sterility. The joint use of partial sterility and dominant conditionally lethal genes has considerable merit if full sterility cannot be induced without serious adverse effects on the release strain. However, release ratios considerably higher than 9:1 are needed to adequately infuse the desired germplasm since this process is impeded by the partial sterility. Obviously, the joint use of partial sterility and conditionally lethal genes is considerably less efficient in the amount of suppression per insect released than the joint use of conventional methods and the release of fully fertile insects with conditional lethals. However, there may be circumstances in which the actual cost of a program which relies on conventional methods to hold the population in check may be considerably higher than a program which relies on partial sterility to suppress population growth while conditional lethals are being infiltrated (*18, pp. 65-79*).

TABLE 11.—*Genotypic trends when a native population (aabb) of 1,000 males and 1,000 females is overflooded in the parental, $F_1$, and $F_2$ generations with insects homozygous for two dominant conditionally lethal genes (AABB). In one model each release consists of 9,000 males only, and in the other each release consists of 9,000 males and 9,000 females*[1]

| Genotype | Males only released, generation | | | | Males and females released, generation | | | |
|---|---|---|---|---|---|---|---|---|
| | $F_1$ | $F_2$ | $F_3$ | $F_4$ | $F_1$ | $F_2$ | $F_3$ | $F_4$ |
| aabb | 200 | 21 | 3 | 3 | 20 | 0 | 0 | 0 |
| Aabb | 0 | 29 | 11 | 27 | 0 | 0 | 0 | 0 |
| AAbb | 0 | 10 | 10 | 54 | 0 | 0 | 0 | 0 |
| aaBb | 0 | 29 | 11 | 27 | 0 | 0 | 0 | 0 |
| AaBb | 1,800 | 635 | 278 | 213 | 360 | 24 | 1 | 1 |
| AABb | 0 | 415 | 489 | 415 | 0 | 19 | 3 | 4 |
| aaBb | 0 | 10 | 10 | 54 | 0 | 0 | 0 | 0 |
| AaBB | 0 | 225 | 489 | 415 | 0 | 19 | 3 | 4 |
| AABB | 0 | 215 | 699 | 791 | 1,620 | 1,937 | 1,992 | 1,991 |
| Total | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 | 2,000 |

[1] The population is held static by conventional means.

## Population Suppression with Recessive Lethal Mutations

In the context of this paper, recessive lethal mutations are expressed in all homozygotes (penetrance and expressivity are assumed to be identical in all individuals). By contrast, recessive conditionally lethal mutations are expressed in homozygotes only under restrictive conditions. For example, winglessness would be lethal to all floodwater mosquitoes with the trait whereas the inability to diapause would be lethal to floodwater mosquitoes only during dry seasons or during winter months. Thus, winglessness would be a lethal trait and nondiapause would be a conditionally lethal trait.

The use of recessive lethals for suppressing pest populations has been considered by several authors (*20, 23, 28*). Indeed, McDonald (*28*) constituted such strains of house flies and studied their performance in population cages.

Recessive lethals can be introduced into a pest population by releasing individuals heterozygous for the lethal genes. We considered the impact of releasing 9,000 males heterozygous for 4 monofactorial lethals (AaBbDdEe) into a native population

(AABBDDEE) of 1,000 males and 1,000 females. The releases have no impact on the rate of increase of the parental generation (fivefold). However, the rates of increase of the $F_1$ generations are reduced to a 3.96-fold and 3.26-fold for one release and for multiple releases respectively. The fold of increase of the $F_2$ is 4.14 and 3.56 for one release and for multiple releases respectively; and the respective values are 4.46 and 4.07 for the F generation. Thus, the release of males with four recessive lethals cannot prevent an increase of population whose intrinsic rate of increase is fivefold.

The maximum impact from recessive lethals can be deduced by considering the consequences of matings between heterozygotes. If the release strain is heterozygous for one recessive lethal gene (Aa), then one-fourth of the progeny of a mating of two heterozygous individuals would die and three-fourths would survive. Further, if the release strain was heterozygous for two independently segregating recessive lethal genes (AaBb) and if two heterozygous individuals mated, then the chance that an individual offspring would not be homozygous for the first recessive lethal would be three-fourths, and the chance that it would not be homozygous for the second recessive lethal would be three-fourths. Thus, the chance that the offspring would not be homozygous for either recessive lethal would be $^3/_4 \times {}^3/_4 = {}^9/_{16}$. In general, the number of surviving progeny from the mating of two heterozygotes is given by $(^3/_4)^n$, where $n$ is the number of independently segregating recessive lethal genes. Thus, the fraction surviving for 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, and 11 genes is 0.750, 0.563, 0.422, 0.316, 0.237, 0.178, 0.133, 0.100, 0.075, 0.056, and 0.042, respectively. To suppress a population increasing at 5-, 10-, or 20-fold rates, the genetic load must be sufficient to permit no more than 20, 10, and 5 percent survival of the progeny, respectively. Therefore, if large numbers of a strain heterozygous for recessive lethals could be released, the strain would have to be heterozygous for at least 6, 8, or 11 recessive lethal genes to suppress populations increasing at 5-, 10-, or 20-fold rates, respectively. We conclude that the use of recessive lethals for population suppression is a rather weak method.

## Trends of a Population Subjected to the Release of Strains of Translocation Homozygotes

The theoretical efficiency of translocation strains in suppressing populations is extremely high relative to most other genetic mechanisms because maximal suppression and maximal stability of

the translocation when introduced into a wild population occurs when the released strain and the wild population are in a 1:1 ratio. Further, the translocation is passed from generation to generation, and it continues to exert an effect on the population until it is eliminated through genetic drift.

To make a theoretical appraisal of the requirements and potential of translocation strains in population suppression, we erected a deterministic model using the following assumptions:

1. The target population consists of 1 million insects with a 1:1 sex ratio.
2. The target population is overflooded once in the parental generation.
3. The native and release strains are equally fit.
4. The number of each strain released is 1 million (500,000 males and 500,000 females).
5. The heterozygotes produced from all mating combinations are identical in their levels of sterility.
6. All matings yield a number of offspring that is directly proportional to the fraction of euploid gametes produced by each parent.
7. From one to three translocation release strains would be developed and released.

Theoretical population trends were calculated, and some of the data are shown in table 12. Our calculations indicate the following conclusions:

- With the release of a single translocation strain, a static or declining population would be decimated, but not one increasing at a fivefold rate.
- With the release of two translocation strains, a downward trend could be induced in a population increasing at a fivefold rate only if the level of sterility of the heterozygotes would be about 95 percent.
- With the release of three translocation strains, a downward trend could be induced in a population increasing even at a tenfold rate, provided that heterozygotes would be 95 percent sterile. With 80 or 90 percent sterility in heterozygotes, a downward trend could be induced in populations which normally would increase at a fivefold rate.

In practice, supplementary measures, such as insecticide applications, would be required against the parental, or perhaps the $F_1$ generation. In addition, the releases should be timed so that the $F_1$, $F_2$, and $F_3$ generations occur during the most adverse season of the year. In this deterministic model, the ratio of one

TABLE 12.—*Population trends when a native population of 1 million insects (1:1 sex ratio) is overflooded once with 1 or more translocation strains*

| Number of release strains | Percentage of sterility | Rate of increase | Insects in generation | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | Parental | 1 | 2 | 3 | 4 | 5 |
| | | | *Mil.* | *Mil.* | *No.* | *No.* | *No.* | *No.* |
| 1 | 95 | 1 | 1 | 2 | 551,250 | 151,938 | 41,878 | 11,543 |
| 1 | 90 | 1 | 1 | 2 | 605,000 | 183,013 | 55,361 | 16,747 |
| 1 | 80 | 1 | 1 | 2 | 720,000 | 259,200 | 93,312 | 35,592 |
| 2 | 95 | 1 | 1 | 3 | 403,333 | 51,226 | 7,290 | 980 |
| 2 | 95 | 5 | 1 | 15 | 10,083,333 | 6,778,241 | 4,556,484 | 3,062,970 |
| 2 | 90 | 1 | 1 | 3 | 480,000 | 76,800 | 12,288 | 1,966 |
| 2 | 90 | 5 | 1 | 15 | 12,000,000 | 9,600,000 | 7,680,000 | 6,144,000 |
| 2 | 80 | 1 | 1 | 3 | 653,333 | 142,281 | 30,986 | 6,748 |
| 3 | 95 | 1 | 1 | 4 | 330,625 | 27,328 | 2,259 | 187 |
| 3 | 95 | 5 | 1 | 20 | 8,265,625 | 3,416,028 | 1,411,780 | 583,462 |
| 3 | 95 | 10 | 1 | 40 | 33,062,500 | 27,328,223 | 22,588,484 | 18,670,794 |
| 3 | 90 | 1 | 1 | 4 | 422,500 | 44,627 | 4,714 | 498 |
| 3 | 90 | 5 | 1 | 20 | 10,562,500 | 5,578,320 | 2,946,050 | 1,555,883 |
| 3 | 80 | 1 | 1 | 4 | 640,000 | 102,400 | 16,384 | 2,621 |
| 3 | 80 | 5 | 1 | 20 | 16,000,000 | 12,800,000 | 10,240,000 | 8,192,000 |

strain to another would remain constant indefinitely. However, in actual practice, this equilibrium would be upset after several generations, and one strain would completely displace the others (*48*)

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